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Presentation of a Basic Defense Program . . 75
Donald E. Currier and Charles H. Bradford

Disseminated Lupus Erythematosus in the Male 81
Bernard H. Pastor, Norman G. Sloane and Harold L. Goldburgh

Single, Circumscribed, Intrathoracic Densities 85
Hans Abeles and David Ehrlich

The Incidence and Heredity of Muscular Dystrophy. 88
Madelaine Ray Brown

Massachusetts Medical Society 112
Obituary 113
Notes from the Medical Examiner 113
Massachusetts Department of Public Health . 114

Fifty Years of Medical Progress: Medicine as a Science: Obstetrics 91
Frederick C. Irving

Case Records of the Massachusetts General Hospital 101

Editorials:

The Plan 110
Responsibility for Hospital Standardization . . . 110
"The Gift and the Giver" 111
Caudal Appendage 112

Correspondence 115
Book Reviews. 115
Notices 116

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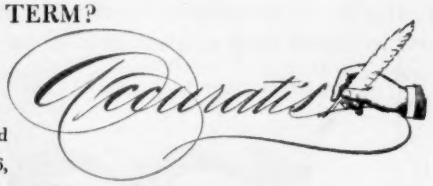

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(1) Schweigert B. S.: Significance of Vitamin B_{12} and Related Factors, J. Am. Dietetic Assoc. *26*:782 (Oct.) 1950.

(2) Lewis, U. J.; Register, U. D.; Thompson, H. T., and Elvehjem, C. A.: Distribution of Vitamin B_{12} in Natural Materials, Proc. Soc. Exper. Biol. & Med. *72*:479 (Nov.) 1949.

(3) Shorb, M. S.: Activity of Vitamin B_{12} for the Growth of Lactobacillus lactis, Science *107*:397 (Apr. 16) 1948.

(4) Hartman, A. M.; Dryden, L. P., and Cary, C. A.: The Role and Sources of Vitamin B_{12}, J. Am. Dietetic Assoc. *25*:929 (Nov.) 1949.

(5) Frost, D. V.; Fricke, H. H., and Spruth, H. C.: Rat Growth Assay for Vitamin B_{12}, Proc. Soc. Exper. Biol. & Med. *72*:102 (Oct.) 1949.

(6) Wetzel, N. C.; Fargo, W. C.; Smith, I. H., and Helikson, J.: Growth Failure in School Children as Associated with Vitamin B_{12} Deficiency—Response to Oral Therapy, Science *110*:651 (Dec. 16) 1949.

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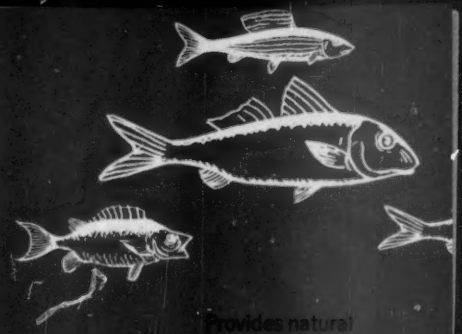

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1. Lewis, J. M., Cohlan, S. Q., and Messina, A.: Pediatrics 5:425, 1950.
2. Lewis, J. M., and Cohlan, S. Q.: Med. Clinics North Amer., March 1950.

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1. *Reeb, B. B., Rohr, J. R., and Colwell, A. R.: Proc. House Staff Dept. Med., Wesley Memorial Hospital, Chicago, Ill, Feb. 6, 1948.*

2. *Rohr, J. H., and Colwell, A. R., Proc. Amer. Diabetes Assn, 8:37, 1948.*

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Neustaedter, T.: Am. J. Obst. & Gynec. 46:530 (Oct.) 1943.

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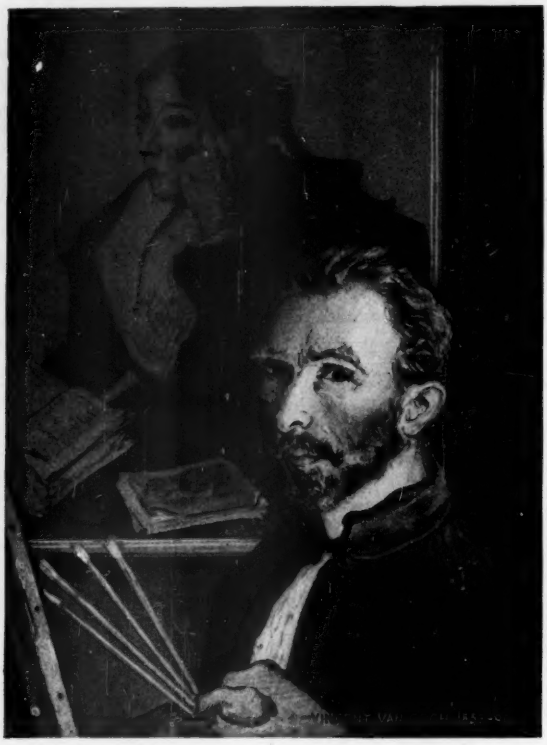

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Volume 244 JANUARY 18, 1951 Number 3

PRESENTATION OF A BASIC DEFENSE PROGRAM

DONALD E. CURRIER, M.D.,* AND CHARLES H. BRADFORD, M.D.†

BOSTON

THE Civil Defense Act of 1950 has created authority for plans and preparations against the emergency of atomic war, but without unstinted public co-operation, no amount of planning can be successful. The Massachusetts Medical Society has already expressed its desire to aid in defense planning through its Committee on Emergency Medical Service. It has also announced its hearty endorsement of the Civil Defense Act and of the appointment of Dr. Vlado A. Getting, Massachusetts Commissioner of Public Health, to take charge as chief of the medical aspects of civil defense. As a guide for promoting further co-operation, it is now time to propose the outline of an over-all program for practical participation in defense plans. No final arrangements can be offered as yet, since many modifications remain to be considered. However, the program submitted here summarizes the essential concepts and offers a general picture of steps that should be developed in the future.

The first and greatest problem of defense against an atomic attack is one of *numbers*. Neither the type of wounds nor the treatment will vary to any great degree from common medical experience. There will be a preponderance of burns, a large number of fractures and traumatic wounds, and a relatively small proportion of radiation-absorption victims. Monitoring the exposed patients will be a technical procedure of importance, but the essential problem that will test the capacity of the medical organization will be the unprecedented number of casualties. To meet this problem, the most important step in planning will be to develop a form of medical teamwork that will reach the highest possible efficiency.

Normally, medicine is the most individualistic of all professions. An atomic emergency will require complete transformation of this approach: doctors will be obliged to treat patients en masse rather than separately. They must abandon the role of independent practitioners and must accept an almost military discipline and control. Such a change cannot be achieved without the preparation of well studied plans. However, if such an organization can be developed, trained and co-ordinated so that each doctor and each hospital will play a part, the present medical personnel of the Commonwealth can undoubtedly handle a major disaster with creditable success, and with tremendous salvaging of life and relief of suffering. This will be an achievement, if it is ever put to the test, that all members of the medical profession will eagerly co-operate in supporting.

*Chairman, Committee on Emergency Medical Service, Massachusetts Medical Society; member, surgical staff, Mount Auburn Hospital, Cambridge.

†Assistant professor of orthopedics, Boston University School of Medicine; member, orthopedic staff, Boston City, Mount Auburn, Malden and Otis General hospitals.

CLASSIFICATION OF EMERGENCIES AND OF TARGET AREAS

Two types of emergency may occur, and defense plans must prepare for both. The first would be a preliminary warning of a definitely threatened attack. This could be by a declaration of war, or by clear signs of immediate outbreak of war, or by an actual act of war occurring outside Massachusetts. In such a case, the Governor would declare the existence of an emergency, and plans for defense would automatically go into operation. The second and more serious type of emergency would be a sneak attack coming without warning and striking within the State. Because such a possibility offers a very real hazard (Pearl Harbor), all preparations must be made for immediate mobilization on the shortest possible notice.

In addition to these two types of emergency, there are three types of territorial zones to consider. The first is what may be called the primary target area — that is, any area of such military or economic importance that it would be singled out for atomic attack at the very start. In Massachusetts, Boston is probably the only target of this sort. A number of areas throughout the State are important enough to attract enemy attacks as secondary targets, if not primary. It must be remembered that Nagasaki was a third-choice selection of the atomic bombing mission that destroyed it. Secondary target areas require plans and preparations similar to those for the primary areas. A third zone could be designated as the reserve area. Here the industrial and economic concentrations would not be great enough to attract either primary or secondary attack though there would always be the pos-

sibility of accidental or missed-target bomb explosions. However, this danger would not justify evacuating such areas, and they should be used as zones for hospital centers around the cities or for base hospitals where evacuees and emergency casualties would be received. It should be a cardinal rule that neither casualties nor evacuees should be moved from one target area to another, whether primary or secondary. Casualties should be routed solely to reserve areas.

Protection Evacuation

As soon as a war emergency develops, the hospitals located within primary target areas should begin evacuating their patients. This movement would be called "protective evacuation." Its purpose would be twofold: to protect the patients from the unnecessary risk of lying helpless in a hospital within a zone where atomic bombing might reasonably be expected and to remove the medical and nursing staffs and essential medical equipment to relative safety, so that they might be ready for the emergency care of heavy casualty lists.

Preparations for this movement would involve three stages:

In the first, the staff of each hospital within a target area should be ready on short notice to discharge as many patients as possible to home care. It would be a matter of judgment whether they were ambulatory enough to return to homes within the target area itself, or whether homes should be found for them in the reserve areas outside the city. In the latter case, homes where patients could be boarded with minimum care could be listed, and such lists made available to the hospital that wanted to use them.

Secondly, each of the major intown hospitals would be assigned in advance to a peripheral base hospital, to which its patients might be evacuated and which the major intown hospital would take over completely in time of emergency. The staff and personnel of the intown hospital would then move into the peripheral hospital and operate it as their own institution, subject to emergency regulations under the Director of Medical Defense. All routine medical or surgical patients would be sent out from the city and treated in this new location.

Thirdly, the intown hospitals would continue to maintain a skeleton force of doctors and nurses, who would carry on an accident and emergency service, but nothing except urgent and immediate surgical or obstetric care would be given here and only overnight wards would be maintained. Thus, essential needs of the city population would be covered without sacrificing the safety of patients or endangering medical personnel more than necessary.

Every industrial center should regard itself as a possible target area, and consequently plans for protective evacuation should be prepared in all secondary as well as in all primary target areas. The secondary areas should await orders from the Civil Defense Authority before carrying out protective evacuation, thus avoiding the confusion of a statewide movement of patients at the very start.

Perimeter Hospitals

During an emergency, medical services of target-area cities would be conducted in a zone at least 10 miles and not more than 40 miles from the center of the city; from 15 to 25 miles would be the optimum radius for this zone. Here the perimeter hospital would be organized, both to meet normal medical requirements of the city and to expand in case of disaster. The hospitals forming this perimeter organization would be composed of three principal types.

Base Hospitals

The major intown hospitals that had moved out under the policy of protective evacuation would be formed into a circle of base hospitals on the perimeter. Their regular staffs would move with them, and they would function on the perimeter, just as they did in the city. They would maintain an emergency staff of their own doctors and of doctors from smaller hospitals who might be affiliated with them during an emergency. This emergency staff would be prepared to answer any sudden call in case of disaster, and the hospital buildings and facilities would be prepared to accommodate 1000 to 3000 emergency casualties in each base hospital. Obviously, each hospital would need to be equipped in readiness for large-scale shock treatments as well as heavy operating-room schedules, burn treatments and fracture treatments. Each hospital would also need to be affiliated with a blood bank and adequate laboratory service. Short-term patients might be kept in these base hospitals until ready to be discharged home; when facilities were overtaxed, such patients might be further evacuated to distant centers or out of the State. Long-term patients would be evacuated as fast as they got over shock and could be made transportable. A national air-transport service for casualties would be of the greatest value. The real service of the base hospital in case of disaster would be to carry the bulk of the worst surgical emergencies, as well as to give shelter to patients whose condition did not justify further evacuation.

The staffs of the base hospitals would include highly-trained specialists in all the surgical branches. Thus, each of these hospitals should organize specialty teams in neurosurgery and plastic, thoracic, genitourinary and orthopedic surgery. The tendency to overspecialization must be avoided, and the base hospitals would need to guard against

wasteful delays of specialization. Long-term procedures requiring repeated operations, such as often occur in plastic or orthopedic work, should be reserved for national centers in the safer interior of the country. The perimeter zones, however, would present many specialized problems, which could be met by programs of specialist consultation in the base hospitals.

Local Hospitals

A strong reinforcement to the base hospitals would be furnished by expansion of the local hospitals already located in the perimeter zone. Every town or community already has such a hospital with a staff that is well qualified for any form of major surgery and with fully equipped buildings, operating rooms, laboratories and wards. With such a nucleus, these hospitals would serve as excellent centers capable of expanding rapidly to three or four times their normal bed capacity. The Concord Plan* represents such an organization. Their staffs should make plans in accord with a maximum load assigned to them. Obviously, their regular buildings are not adequate for such an expansion, but they can have in readiness a plan and equipment for utilizing neighboring schools, gymnasiums or public buildings wherever heating, plumbing and floor space would permit concentrating a large number of cots. This would provide for a large overflow of patients. It would be important to group as many together as possible in order to save the time of the nursing and medical staffs, and it would also be important to establish such auxiliary wards reasonably close to the parent institution.

A second means of expansion would be by providing accommodations for patients who had suffered lighter casualties that permitted them to be ambulatory. Since many casualties would come from bombed-out homes, this group of cases might be unusually large. They could be concentrated in local hotels, which could be commandeered for this purpose, or arrangements could be made to improvise for them in large public buildings. In summer, they could be sheltered in tents. Rolling kitchens could supply meals for these ambulatory patients. Their treatments could be conducted on an outpatient basis either at the hospital itself or at the improvised center.

Mobile Hospitals

A third reinforcement to the hospital perimeter would be furnished by mobile hospital units. These could be organized on a basis similar to that of the portable surgical hospitals of the last war. They should be equipped in advance so as to make them completely mobile, with cots, surgical equipment, x-ray apparatus, supplies and a staff of doctors, nurses, orderlies and technicians to meet any surgical emergency within their sphere. They should have a bed capacity for 100 patients, but their purpose should be more to treat and evacuate casualties than to hold them. They could be moved in close to a bombed-out area, and could be set up beside the main highways in association with casualty assembly points or sorting stations. Here, their help would be invaluable both in sorting the wounded and in giving immediate care to minor injuries, thereby relieving the burden of further evacuation and saving the perimeter hospitals from an unnecessary overload of patients. A second function for these mobile units could be to supplement the other perimeter hospitals. In case the local or base hospitals were overwhelmed with a load of major surgical emergencies, one or two mobile units could move in close beside them and help handle the overflow until the peak had passed. Thus, without any very great difficulty, an important auxiliary surgical service could be instituted. The tables of organization and details of planning would have to be left to special study.

*McDonald, F. C., and Dalrymple, S. C. Function of suburban hospital in civilian defense: Concord plan. *New Eng. J. Med.* (in press).

Hospital Allocation

The foremost difficulty in organizing the hospital perimeter lies in the lack of an adequate number of hospital buildings around the larger cities. The most suitable institutions for conversion in time of emergency would be the mental hospitals. These could be transformed into large, active base hospitals with relative ease, if their patients could be provided for. Perhaps some hardship would be involved in transferring these patients, but this would be compensated for by the urgency of the need in times of disaster. It might be possible to equip boarding schools or even large resort hotels to receive the mental patients; or they might be moved temporarily out of the State. As a group, they are rather easily transportable, and with relatively little danger to themselves. The essential part of any plan for transferring the mental patients would be to carry it out as fast as possible.

A second possibility for allocating base hospitals would be through use of existing military hospitals in the perimeter zone. This would, of course, require clearance with the Defense Department, and it might meet the objection that such hospitals are necessary for patients in the armed forces. Actually, however, the civilian need would be far more urgent, particularly in the neighborhood of the great, target-area cities. The country as a whole offers huge reserve areas, neighboring to smaller communities, far removed from atomic targets, where service hospitals could and should be established. In this respect, it would seem that the medical services of the Department of Defense could aid immensely if the entire policy of hospitalization were revamped.

Still a third means of establishing base-hospital centers lies in utilization of Veteran Administration hospitals outside the city limits. Although these institutions are already in full operation, their bed capacity could rapidly be expanded by hasty construction of additional wards around them. Furthermore, their elective operative schedules could be terminated so that all their beds would be free for emergency service.

Provision for a Sneak Attack

In case of the very real possibility of a sudden sneak attack with atomic bombs, the disaster that followed would be the hardest problem for civil defense to meet. The very fact that unexpected disasters may occur should stimulate defense planning to make ready as much of the medical program as can be prepared in advance. Even without base hospitals on the perimeter, it would be possible to handle large numbers of casualties through the other agencies already described. A complete plan for location and for staffing and equipping the casualty clearing stations could be in readiness at all times; likewise, plans and equipment should be ready for emergency expansion of local hospitals in the perimeter zone. Mobile hospitals from each medical district could be kept on the alert for emergency calls, and an ambulance service could be prepared to move out in response to an immediate summons. The blood banks and laboratory services could also be prepared to go into action promptly. If an atomic bomb destroyed the intown hospitals, as would be expected, any doctors or nurses who survived could be assigned to the staffs of local hospitals on the perimeter, where medical support would be desperately needed.

A second lesson to be learned from the dire possibilities of sudden atomic-bomb attacks would be to teach one the need of immediate protective evacuation of target areas in the event that the first attack should fall outside Massachusetts and thus give warning of imminent danger. Plans should therefore be in readiness for conversion of mental hospitals with the utmost speed and for transfer of patients in the shortest possible time. Delay of a week or even a few days would simply be an invitation to the enemy to come again with return attacks and fresh disaster. The speed with which civil defense can mobilize will depend in great measure on the thoroughness of its preparation. Delays of any sort would bring untold suffering and would mean the unnecessary loss of thousands of lives.

Casualty Clearance

The shortage of medical personnel in comparison to the needs of a disaster makes it unwise to assign doctors or nurses to the rescue of bomb victims or to collection or transportation of the wounded out of the target zone. This initial stage of casualty clearance would therefore depend entirely on nonmedical teams. Radiologic monitoring of the bombed areas and of the victims would be necessary in regions near the center of the burst, but this work could be conducted by technically trained officers without any medical background. Only the most elementary first-aid measures would apply in the initial phase of clearance, and any form of elaborate splinting, bandaging or dressing should be avoided. In Britain, during the blitz, it was found that first-aid treatments tended to delay patients on their way to more adequate medical care. Therefore, it was believed that no aid at all would be better than too much. Obviously, hemorrhage should be stopped at the earliest stage of rescue work, and before transportation. Secondly, broken limbs should be protected with rudimentary splints. Thirdly, all rescue workers or first-aid teams should be carefully instructed regarding movement of patients with fractures of the vertebras. Possibly, these three steps of treatment or protection are the only points that should be allowed to delay the patients at all in the primary stage.

Under atomic-bomb conditions, with collapse of stone buildings over a wide area, carriage of patients on stretchers by hand might be necessary on a large scale and for relatively long distances. Ambulances improvised from private cars or trucks would penetrate as far into the bombed area as possible so as to meet the litter bearers. Each section of the city would need an advance plan for the location of assembly points where the litter cases could be gathered. From these primary assembly points, the patient should be carried to pre-arranged casualty clearing stations, staffed by teams of doctors, nurses and aides. The sites of these clearing stations should be selected with several points in mind:

They should be close enough in the city to be as readily available as possible, without being exposed to a central burst over the target area — that is, from 2 to 6 miles from the center of the city.

They should be situated along a natural "line of drift of the wounded" — that is, on a main street going out of town reasonably close to a main crossroads.

They should be located in a large, ground-floor building where there is plenty of room for hundreds of litter cases to be laid out and where ambulance traffic could move with ease.

A ring of such casualty clearing stations should be designated in advance around the city. The stations should be organized in depth, so that if the inner stations were rendered unserviceable, others would be available a little farther in the rear, but still not too far away. There should not be too many stations, since wide dispersion of the wounded would delay them on their way to the perimeter

hospital and would add to the difficulties of ambulance convoys. Possible twenty-five to thirty-five such stations around the city would be adequate.

The casualty clearing stations should serve as the only stops between collecting patients at the scene of injury and delivering them at the perimeter hospitals. The clearing station should be particularly well equipped for monitoring patients against atomic exposure, for primary treatment of the less severe burns, for administering plasma when indicated and for sorting out the moribund wounded for whom further evacuation was either useless or too dangerous to be worth the risk. Except for the above purposes, the main function of the clearing station would be to evacuate the wounded as fast as possible. Ambulance companies should be assigned to these stations so that movement of patients could be expedited as much as possible. There should also be a control officer at each station, who would be in communication with the control office. By such a means of control, it would be possible to distribute the load of casualties and obtain maximum benefit of all the services in the rear. It would also be possible to route certain types of cases to special centers, if it seemed advisable to do so. Patients with radioactive injuries might be filtered out of the casualties at these points, and either treated at the station or isolated in special centers. There might also be special selection of burn cases or of neurosurgical cases. The control officer would be informed of general policies in these matters, and would be in a position to carry them out at the clearing station rather than having a rerouting and a second ambulance trip beginning at the perimeter hospital.

Mobile hospital units might prove of great help if they could be moved up promptly after a disaster to serve close beside the casualty clearing stations or in immediate conjunction with them. They could relieve a large part of the load of less seriously wounded, who would only clog up hospitals if evacuated farther back. They could also aid in shock treatment of the patient with extremely severe injuries, many of whom might be saved, even though apparently moribund at this point. Shortly after a disaster, the stations where the greatest number of wounded were accumulating would soon become apparent, and mobile hospitals would be particularly useful if moved in to such points.

District Defense Committee Planning

In order to make the plans already outlined workable, the defense committees of the district medical societies and the state society should make preparations in detail. There is not space here for a complete summary of all phases of planning, but in general, four aspects of special importance deserve emphasis.

Emergency Assignments

Every available doctor in each district should be given an emergency assignment. This would necessitate the compilation of an "alarm list" in the district society headquarters, where the names of all doctors registered in the district would be recorded, together with the duties to which they had been assigned. Doctors subject to call in the armed forces should be marked as unavailable in this emergency listing. Many doctors maintain offices in two different districts. Possible duplication of confusion from this source should be eliminated in preparing the alarm lists. When finally completed, these lists should have an assignment for each doctor. Individual summonses of doctors at the time of an emergency should not be necessary. If every doctor knew in advance where he was expected to go, he could be relied upon to report at his post as soon as any general emergency alarm was issued.

Staffing of Casualty Clearing Stations and Special Training

Each district in or adjoining a target area should arrange for assignment of an adequate staff to the casualty clearing stations designated by the Director of Medical Defense within the district. It should be the responsibility of the Defense Committee to see that the clearing-station teams were properly in readiness at all times and also that they were well trained in their duties, and had a clear grasp of the plans of evacuation. Each clearing-station team should have a chief officer and a deputy chief who would undertake to train the group and to be responsible for their equipment, their familiarity with the location of their station and their availability in case of emergency.

Hospital Preparations

Every local hospital in the State would be affected by defense planning, and therefore each hospital should form a defense committee to co-operate with the district medical society and with the office of the Director of Medical Defense. These hospital committees should assume responsibility for assignment of their own staffs. It would probably be advisable to organize relays of surgeons on day and night operating teams, as well as organizing specialty teams for burns, shock, fractures, abdominal and chest surgery and head injuries. Special staff assignments for the admitting rooms and for sorting of patients at the time of their first arrival should be provided for.

A second responsibility of the hospital defense committee should be to plan for expanding their facilities, as already outlined above. The only means for meeting the terrific overload of disaster casualties would lie in the efficiency with which hospitals could expand. Of course, every expanded facility would need to be covered by the assignment

of medical personnel from the hospital staff. Cases would require supervision in the new wards, treatments would have to be given, outpatient service might be necessary, and minor or major surgery might be called for.

The defense committee of the intown hospitals within the target areas would be faced with the need for planning in two directions. One would be to prepare for immediate evacuation of their hospitals in case of an emergency warning. The second would be to form a co-ordinating committee to prepare for staffing and taking over the base hospital to which they had been assigned. As mentioned above, this change-over to a perimeter location must be carried out with the greatest possible speed, and intensive planning for the move would be required in advance.

Mobile Hospitals

Each district should be able to form at least one mobile hospital unit. The district defense committee should take charge of organizing such a unit subject to specifications issued by the Commissioner's office. Such a unit would serve under the leadership of a chief surgeon, who would be assisted by a surgical and a nursing staff together with technicians, orderlies, drivers and mess personnel. The chief surgeon should make himself responsible for the entire unit, including equipment, and should act in the capacity of a commanding officer. Medical districts in the center of primary bombing areas, and possibly in the larger secondary areas, would not be in a position to form mobile hospitals owing to their exposure to attack. This could be remedied by calls to other districts to form more than one mobile hospital if they considered themselves able to do so.

Ambulance Service

The formation of an ambulance service does not lie directly in the sphere of medical preparations, but it is important to point out in this outline that such a service is absolutely essential to the success of the entire plan. There is danger that the need for this very vital service, not being a medical function, will be overlooked. Because of its essential nature as an integral part of the whole medical program, it is urgently recommended that very special planning should be devoted to creating an ambulance service adequate to meet the emergency needs of the State. After such a service had been organized, it should be subjected to test mobilization on short notice. In times of disaster it should become an active part of the medical defense program and should serve under the personal orders of the Director of Medical Defense.

Command Function

Efficient management of such a large organization as an emergency medical service would require a great concentration of authority in the hands of one man — the chief medical officer of the State. He would select his own deputies and representatives, who would form his staff. Because the benefits of this centralized control would affect the medical profession directly, a further word of explanation seems warranted.

The office of the chief medical officer derives its authority from the Commissioner of Civil Defense, who is appointed by the Governor. There is an advisory board appointed by the Governor to confer with the Commissioner, and there is also an advisory committee selected by the medical chief. Through the action of these bodies, the advantage of consultation with groups of experts is combined with a necessary concentration of authority in the hands of one man. The entire framework of the organization is designed solely for emergency needs, and it is believed that the conditions to which it would apply not only justify but demand a dictatorial centralization of power.

During the present planning stage, the function of preparing for an emergency is being actively and energetically carried out by the civil defense authorities of the State. In addition to their own studies, they are giving thoughtful consideration to all suggestions that are offered. The Committee on Emergency Medical Service of the Massachusetts Medical Society has already been invited to participate in this planning, and further co-operation of district defense committees will be welcomed. Even after a final defense plan has been adopted, modifications or improvements may still be considered. The ultimate aim is to develop an organization that can be expanded to a nation-wide scale. It is not necessarily advisable that each state should adopt precisely the same plan. However, it is essential that all plans should be capable of dovetailing in the final pattern of activity.

At a time of actual emergency or disaster all planners will agree upon the need of centralized control. Therefore, under this plan, the chief medical officer would assume charge of all medical personnel and facilities within the State once an emergency had been declared. He or his staff would be prepared to take over the assignment of doctors, the movement of patients, the organization of hospitals, the posting of casualty clearing stations, the work of aid parties, the distribution of medical supplies and all related activities. He would exercise the authority of a commander-in-chief, and it should be understood in advance that any orders from his

office would merit immediate and complete co-operation. Without such centralization of authority, the medical organization could not co-operate efficiently, but with it all agencies of the State could be co-ordinated in a swiftly moving and effective machine. It is anticipated that co-operation of all units would be voluntarily given; but in addition the Civil Defense Act has granted the Commissioner adequate legal powers to enforce his orders if that should be necessary. However, such authority will undoubtedly never be required, for the public realizes the critical dangers of an emergency and knows in advance the degree of co-operation that is expected of it.

DISSEMINATED LUPUS ERYTHEMATOSUS IN THE MALE*

Report of Five Cases

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MOST reports on disseminated lupus erythematosus have considered it to be a diffuse disease of the collagen system occurring almost exclusively in young adult women. Blount and Barrett,[1] reporting a case in a young man, make the observation that the occurrence of this entity in the male is unusual. All the 20 cases reported by Klemperer and his associates[2] were in women, and Baehr and Pollack[3] state that the most striking characteristic of the disease is its predominant occurrence (95 per cent) in young women. However, 3 of the 12 cases reported by Rose and Pillsbury[4] were in males. A somewhat smaller percentage was noted by Fox and Rosahn.[5] In 1943 they reviewed most of the reported cases in an effort to determine the incidence of lymph-node enlargement. Of 277 cases, 228 were in females and 49 in males, an incidence of 17.7 per cent in the latter.

At the Philadelphia General Hospital, the clinical diagnosis of disseminated lupus erythematosus has been made in 36 cases since 1936. Of this number, 10 were in males. This roughly corresponds to the incidence noted by Rose and Pillsbury.[4]

The following cases of 5 male patients with disseminated lupus erythematosus in whom there was pathological confirmation of the diagnosis illustrate some of the clinical and pathological features of the disease.

CASE REPORTS

CASE 1. J. N., a 25-year-old man, was admitted to the service of Dr. H. L. Goldburgh at the Philadelphia General Hospital on October 9, 1948. He had been treated for migratory arthritis involving all the large joints in June, 1948. A diagnosis of rheumatoid arthritis was made, and he was hospitalized because of failure to respond to a 3-month course of gold therapy. There was a history of pulmonary tuberculosis in 1942. The response to rest, salicylates and physiotherapy was satisfactory, and he was discharged on October 23. At this time his pulmonary lesion had been "arrested" for 2 years although pneumothorax was still being maintained.

He was readmitted to the hospital on December 27 because of recurrence of the acute arthralgia. Physical examination revealed a well developed, fairly well nourished man who looked pallid and chronically ill, and who was acutely dyspneic. The temperature was 101° F., and there were signs of consolidation over the right lower lobe. The heart was normal except for a rate of 110 per minute, and the blood pressure was 135 systolic, 95 diastolic. The spleen was enlarged. Pitting edema of the hands and feet was present, and all the joints were swollen, hot and painful upon passive motion. Examination of the blood disclosed a hemoglobin of 10.0 gm., and a white-cell count of 3200, with 86 per cent neutrophils, 12 per cent lymphocytes and 2 per cent monocytes; the hematocrit was 31.2 per cent, and the serum protein 4.9 gm. per 100 cc., with an albumin-globulin ratio of 2.7:2.2. The blood sugar and urea nitrogen were normal. A serologic test for syphilis was negative. Urinalysis demonstrated a specific gravity of 1.025, with a ++++ test for albumin, and the sediment contained 2 to 4 white blood cells and many red blood cells per high-power field; the sedimentation rate was 60 mm. per hour. An x-ray film of the joints was reported to be consistent with the early changes of rheumatoid arthritis.

On January 6, 1949, an acute right-upper-lobe pneumonia developed. Penicillin therapy was begun, with prompt clinical improvement. The temperature fell to normal, but rose to 101°F. as soon as therapy was stopped on January 15. There was no return of joint or chest symptoms at this time, and the principal complaints were weakness and epigastric distress. During the acute febrile episode, a scaly erythematous rash appeared on the upper lip and nasolabial angle, resembling a seborrheic dermatitis.

On January 22 a blood transfusion was given. Two days later, a pneumothorax refill of 250 cc. was administered by the consultant phthisiologist, who believed that active tuberculosis was not present. Several 24-hour sputum examinations for acid-fast bacilli were negative. A blood culture was also negative. The temperature remained elevated. A bone-marrow biopsy was done and showed changes compatible with a "toxic state."

On January 26 the patient complained of diplopia and night sweats. Joint symptoms had subsided. The liver and spleen were much enlarged. He was given a transfusion, which was followed by an attack of dyspnea that subsided promptly with oxygen and sedation. The electrocardiogram, which had been normal in October, now showed flattening of most of the T waves and inversion of the T waves in Leads aVL and CR_4. Fluoroscopy revealed 25 per cent cardiac enlargement and some hilar congestion.

Clinical opinion at this time favored a diagnosis of one of the "collagen" diseases, although direct evidence was lacking. Terminally the picture became more characteristic of acute disseminated lupus erythematosus. There was rather sudden deterioration of the patient's condition on February 7, with chest pain, dyspnea, diarrhea and temperature elevation to 104°F. Despite oxygen, penicillin and other supportive measures he became steadily worse. On February 10 swelling of the throat, mouth and tongue developed, making swallowing difficult. He was depressed and repeatedly expressed a desire

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to die. Multiple small ecchymotic areas on the hands, arms and legs and hemorrhages into the tongue and buccal mucosa appeared. There were acute ulcerations in the mouth and under the tongue. The swelling of the lips and tongue increased so much that respiration was difficult and preparations for tracheotomy were made, but complete obstruction did not develop. The patient died later the same day.

At autopsy* the body was that of a well nourished man. There were acute exudative lesions about the oral cavity, acute glossitis with ulceration and multiple hemorrhagic lesions of the skin. The heart was hypertrophied and dilated. Typical Libman-Sacks endocarditis of the tricuspid valve, chronic pericarditis and a myocarditis with Aschoff-like nodules were present. The lungs gave evidence of healed tuberculosis, nonspecific pleuritis, congestion and edema. There was splenomegaly and hepatomegaly, with marked perisplenitis and perihepatitis. The kidneys showed marked glomerular changes typical of lupus. The tongue revealed hyperkeratosis, acantholysis and vesicle formation in the epithelial layer, and intense congestion and acute inflammation of the papillary and muscle layers. The skeletal muscle disclosed edema, chronic myositis and foci of collagen necrosis. The pathological diagnosis was acute disseminated lupus erythematosus.

CASE 2. H. C., a 38-year-old man, was admitted to the service of Dr. D. N. Kremer at the Philadelphia General Hospital on December 24, 1939, with a history of migratory joint pains and swelling of 3 weeks' duration, involving the ankle, left knee, right elbow and both hands. He had a continuous low-grade fever. One week before admission a rash appeared on the hands, face and scalp. For 5 years he had had a chronic cough with pain in the left side of the chest and occasional hemoptysis. There had been a 15-pound weight loss. The past medical history was not otherwise significant.

On admission he did not appear very ill. Small macular lesions were present on both hands. These were circular, flat and reddened, and each was in the center of a small area of alopecia. A red, indurated lesion was present between the bridge of the nose and the inner canthus of the right eye. There was slight generalized lymphadenopathy.

An electrocardiogram was normal. X-ray study of the chest revealed some free fluid at the left base. Urinalysis, blood sugar, blood urea nitrogen and urea clearance were normal. Examination of the blood showed a red-cell count of 3,400,000 with a hemoglobin of 10 gm., and a white-cell count of 5800 with 20 per cent nonsegmented and 32 per cent segmented neutrophils, 42 per cent lymphocytes, 4 per cent monocytes and 2 per cent eosinophils. The blood Kahn reaction was +++. The sedimentation rate was elevated on December 24, 1939, and was normal on January 11, 1940.

A dermatologic consultant at first thought that the lesions on the face were lupus erythematosus and the hand lesions papulonecrotic tuberculids, but changed his diagnosis to erythema elevatum diutinum, and interpreted a biopsy as compatible with this later diagnosis. Antisyphilitic therapy with bismuth was started on January 15, and the patient was discharged to the clinic.

He was readmitted to the hospital on February 2 for joint pain and was discharged "improved" 1 week later. He was again admitted on July 14, 1944, acutely ill, dyspneic and confused. He had bilateral pleural effusion and edema of the lower extremities. There were macular reddened plaques in the "butterfly" area of the face. These lesions showed a tendency to coalesce, and were atrophic. Dusky-purplish, atrophic, deep-seated macular lesions were present on the palmar surface of the fingers.

An x-ray film of the chest showed effusion at the bases. Examination of the blood disclosed a red-cell count of 3,200,000, with a hemoglobin of 10.5 gm. per 100 cc., and a white-cell count of 5000, with 22 per cent segmented and 40 per cent nonsegmented neutrophils and 38 per cent lymphocytes. Urinalysis demonstrated a ++++ test for albumin, with granular casts and numerous red and white blood cells in the sediment. The blood urea nitrogen was 15 mg. per 100 cc. The blood Wassermann reaction was +++, and the blood Kline reaction ++. The serum protein was 5.6 gm. per 100 cc., with an albumin-globulin ratio of 1:1. Another white-cell count was 2300, with 34 per cent segmented and 38 per cent nonsegmented neutrophils, 22 per cent lymphocytes and 6 per cent monocytes.

*Performed by Dr. S. Swift.

On admission the temperature was only slightly elevated. It later rose as high as 104°F. Repeated thoracenteses were necessary.

Penicillin was given in doses of 100,000 units per day. At first there was marked spread of the skin lesions, but during the 2nd week they disappeared almost completely, the temperature returned to normal, and the pleural effusion receded spontaneously.

The dermatologic consultant believed that the lesions were those of disseminated lupus erythematosus, and he elicited the history that they were made worse by exposure to sunlight. A biopsy was taken, and a pathologist also reviewed the old slides. The pathologist's report was as follows:

> Microscopical examination of the skin reveals essentially the same picture as that of the former biopsy. The corium adjacent to the epidermis shows fibrinoid degeneration and necrosis, and there are infiltrations with round cells and a few polymorphonuclear leukocytes, especially around the small blood vessels. The epidermis proper is necrotic in places, the basal layer being more severely involved than the other layers. There is nothing to suggest granuloma annulare. The diagnosis is lupus erythematosus disseminatus.

The patient was discharged to the clinic on October 3. The subsequent course was progressively downhill, with several readmissions for pleural effusion requiring repeated thoracenteses, cardiac decompensation and one bout of pneumonia. He died on January 16, 1945.

Permission for autopsy was refused.

CASE 3. J. C., a 27-year-old grocery clerk, had been a patient at another hospital from March 20 to April 10, 1943, where a diagnosis of early infectious arthritis was made. The sedimentation rate was moderately elevated and an electrocardiogram was reported to be within normal limits. A routine blood count and urinalysis were normal, and a serologic test for syphilis was negative. He was readmitted to the same hospital on July 21 with pain and stiffness in the elbows, knees and shoulders and with rather marked anorexia. There was an erythematous, papular, confluent rash over the forehead, the "butterfly" area of the face, the exposed area of the chest and both deltoid regions. There was slight anemia. The urinalysis, blood urea nitrogen and urea clearance were normal. An Addis count, however, revealed definitely abnormal excretion of albumin and casts. Minimal ultraviolet radiation over the back produced an exacerbation of the joint symptoms, a temperature elevation and a white-cell count of 10,000.

He was admitted to the service of Dr. F. Baumann at the Philadelphia General Hospital on October 11, with a history of transient attacks of polyarthritis with some joint swelling for the past year. Ankle edema, ascites, dyspnea and cough developed in August. There had been a 20-pound weight loss. During the week before admission, nausea, vomiting, shaking and chills developed. There were periods of confusion.

On physical examination, the patient appeared thin, dyspneic, and quite pale. The temperature was 98.6°F., the pulse 74, and the respirations 20 per minute; the blood pressure was 112/80. An erythematous rash was present on the chest, and there was a shallow bleeding ulcer on the lower lip. There were rales at both bases. The heart was enlarged, and a mitral systolic murmur was heard. There was a moderate degree of ascites, with a tender liver edge two finger-breadths below the costal margin. Edema of the feet, ankles, thighs and scrotum was present. On admission, the red-cell count was 1,700,000, with a hemoglobin of less than 7.5 gm., and a white-cell count of 7100, with 68 per cent neutrophils, 29 per cent lymphocytes and 3 per cent eosinophils. Urinalysis was normal. The blood sugar was 70 mg., and the blood urea nitrogen 125 mg. per 100 cc. The prothrombin time was 57 per cent of normal, and a serologic test for syphilis was negative. The sedimentation rate was 23 mm. in 15 minutes.

The patient was given five blood transfusions, Aminoids intravenously and other supportive therapy, without much benefit. Abdominal distention developed. During the hospital stay there was a constant low-grade fever, with occasional rises to 101 or 102°F. An electrocardiogram was within normal limits. White-cell counts ranged from 6600 to 8600.

The blood urea nitrogen fell to 70 mg. per 100 cc. on October 26 but subsequently rose to 90 mg. The urine specific gravity ranged from 1.012 to 1.017, always with ++++ tests for albumin and granular casts in the sediment. Several serum protein determinations averaged 3.5 gm. per 100 cc., with an albumin-globulin ratio of 1.3.2.2. Chloride determinations were in the range of 600 to 640 mg. per 100 cc., and the carbon dioxide combining power on November 20 was 27 vol. per cent. The serum calcium was 7.8 mg. per 100 cc.

During the last week of his illness, the patient lapsed into periods of unconsciousness lasting several minutes each. On November 22 he began to regurgitate watery, brown material. He became progressively weaker and died the following day.

At autopsy* there was adhesive pleuritis and pericarditis. A chronic peritonitis was present, with marked perihepatitis and perisplenitis. The heart was hypertrophied and dilated, and there was inflammation of the mitral and tricuspid valves. Fibrinoid degeneration of the corium of the skin and healing mucosal lesions on the tongue suggestive of lupus were found. In the kidneys, a fibrinoid glomerulonephrosis with characteristic wire looping was present. The pathological diagnosis was disseminated lupus erythematosus.

Case 4. W. H., a 12-year-old Negro schoolboy, was admitted to the service of Dr. P. György at the Philadelphia General Hospital on January 1, 1949. He had been in excellent health until December 25, 1948, when fever, malaise, arthralgia and myalgia had developed. The pain became so severe that he was unable to move, and he had a persistent headache. Several episodes of epistaxis occurred. "Sores" on the lips developed, and a skin lesion, which had been present on the face for about a month, became worse. He complained of substernal pain, shortness of breath and palpitation, but had no cough or hemoptysis. The diagnosis on admission to the hospital was acute rheumatic fever.

Physical examination showed a moderately well developed but poorly nourished and acutely ill boy. The blood pressure was 108/90. The temperature was 104°F., the pulse 100, and the respirations 24. Conjunctivitis was present, the lips were swollen, and there was dried fresh blood around the nares. There were discrete enlarged lymph nodes in the anterior cervical and axillary regions. No abnormal cardiac findings were noted. Some generalized abdominal tenderness, most marked over the spleen, was present. Numerous deeply pigmented papular lesions of varying size were present on the face, and there were many irregular scars on the forehead and left cheek.

Examination of the blood demonstrated a hemoglobin ranging from 9.6 to 12.8 gm. and a persistent leukopenia (with a white-cell count of 1800 to 5750), most of the counts being around 3000, but the differential count was not abnormal. The erythrocyte sedimentation rate was 86 mm. in 1 hour. The platelet count was 80,000 direct and 92,000 indirect. No sickling was present. The prothrombin time was normal. Serum protein determinations were 7.9 gm. per 100 cc., with an albumin-globulin ratio of 3.4:4.5 on one occasion, and 6.4 gm., with a ratio of 2.8:3.6 on another. The blood urea nitrogen ranged from 9 to 26 mg. per 100 cc., the blood sugar was 96 mg. per 100 cc., and the chloride was 120.4 milliequiv. per liter. Blood cultures were persistently negative. The urine showed persistent albumin, red blood cells, white blood cells and casts. The bone marrow was normal, and no target cells or cells characteristic of lupus erythematosus were seen. An x-ray film of the chest and an electrocardiogram were normal.

The patient's temperature varied from 103 to 104°F., and he had a fulminating downhill course. The face lesions spread and became ulcerative. Cardiac decompensation developed. Treatment consisted of bed rest, penicillin, Benadryl, streptomycin and blood transfusions. He was incontinent of urine and feces, lapsed into coma and died on January 25.

At autopsy† the body was that of a poorly nourished boy with a generalized skin eruption most marked on the face and trunk, and involving the mucous membranes of the mouth. There was fibrinous pleuritis and hypostatic pneumonia, but no pleural effusion. Moderate lymphadenopathy of mediastinal and mesenteric nodes was present. There was focal fatty dystrophy of the heart, with moderate epicardial fibrosis. A minimal degree of "wire looping" in the kidneys was noted. The pathological diagnosis was disseminated lupus erythematosus.

Case 5. J. B., a 30-year-old Negro, was admitted to the service of Dr. D. N. Kremer at the Philadelphia General Hospital on September 30, 1949. He had complained of migratory arthralgia since 1947. All joints had been involved at various times. Temperature elevation and sedimentation rates of 25 to 30 mm. per hour were associated with the attacks of arthritis. Smears of a urethral discharge and complement-fixation tests for gonococci were consistently negative, as was the serologic test for syphilis. On two previous hospital admissions in 1949 a diagnosis of arthritis of unknown etiology had been made. Since the last hospitalization he had been unable to work because of joint swelling. Urethral discharge continued.

On physical examination the temperature was 100°F., the pulse 88, and the respirations 24. The blood pressure was 120/70. There was increased warmth of the right shoulder, left elbow and wrist and right knee. There was fluid in the right knee joint. A positive smear for gonococci was obtained from the urethral discharge, and a diagnosis of gonococcal arthritis was made. Because of the long duration and chronicity of the arthritis, fever therapy was given in addition to penicillin. After the second treatment, chest pain and a pericardial friction rub developed. Fever therapy was therefore discontinued. The cardiac silhouette became progressively enlarged, and cardiac decompensation developed on October 29. Positive blood cultures for *Streptococcus viridans* were obtained on October 31 and November 19. Bone-marrow biopsy revealed 6 per cent plasma cells and increased cellularity, but no cells typical of lupus erythematosus were noted. The subsequent course, with bronchopneumonia and recurrent thrombophlebitis, was not altered by streptomycin therapy. The patient lapsed into coma and died on January 17.

At autopsy‡ the body was emaciated. There was diffuse fibrinous pleuritis, pericarditis and a suppurative pelvic peritonitis. Marked fibrinous capsulitis of the liver and spleen was present. The heart was moderately dilated, and there was typical Libman-Sacks endocarditis of the tricuspid and mitral valves. The lungs showed diffuse nonspecific pneumonia, congestion and edema. There was moderate enlargement of the cervical, axillary and hilar lymph nodes. Pyogenic arthritis was present. The kidneys showed glomerular changes consistent with lupus erythematosus. The joints demonstrated proliferative synovitis, and the skeletal muscle chronic focal myositis. The pathological diagnosis was disseminated lupus erythematosus and pneumonia.

Discussion

Attempts have been made to classify lupus erythematosus as acute disseminated, subacute disseminated, chronic disseminated and chronic discoid (localized) forms. The clearest distinction has been between the localized and disseminated types. Differentiation of acute and subacute types is based merely on the severity of the constitutional symptoms and the rapidity of the course.[6] Graffin and his co-workers[7] have recently reviewed the clinical and pathological features of this syndrome.

The most frequently reported clinical and laboratory features are fever, toxicity, arthralgia, acute arthritis, purpura, cardiac murmurs, tachycardia, lymphadenopathy, polyserositis and cutaneous lesions, associated with anemia, leukopenia, thrombocytopenia, albuminuria, microscopical hematuria, reversal of the albumin-globulin ratio, false-positive serologic tests for syphilis and the absence of bacteremia.

The cutaneous eruption may vary from a faint, nonspecific erythema to the more characteristic

*Performed by Dr. A. Valdez-Dapena.

†Performed by Dr. M. Schick.

‡Performed by Dr. S. Kustermann.

discoid lesion or from an erythematous infiltrative plaque with dilated hair follicles containing white scales to hyperpigmented or depigmented atrophic scars. The lesions may involve the "butterfly" area of the face, the scalp, ears, upper trunk, fingers, toes and oral mucosa.

The pathological features of the cutaneous lesions are believed by Montgomery[8] to be typical and diagnostic, and he describes them in some detail. Klemperer[2] interprets the earliest cutaneous change as a homogenization of collagen fibrils in the most superficial layer of the corium. Fibrinoid degeneration occurs next, and in the most advanced lesion, the clumped fibrinoid masses break up and become basophilic. Secondary degenerative changes take place in the epidermis. Vacuolization occurs and may progress to necrosis of the basal cell layer.

The heart is involved in a large percentage of cases. The fully developed endocardial lesions, first described by Libman and Sacks,[9] are, according to Klemperer,[2] pathognomonic. The vegetations are dry, granular, tawny or pink, and vary from 1 to 4 mm. in size. The lesions are on the valves, in the sinuses, on the arterial side of the semilunar cusps, on the endocardium of the ventricular outflow tract, on the chordae tendineae and papillary muscles, and in the angles between the auriculoventricular valves and the mural endocardium. In addition to the endocardial lesions, which are small, swollen masses of altered ground substance, there are similar changes in the collagen of the deeper endocardial layers and the vascular and supporting tissues of the myocardium. Secondary changes are found in the muscle fibers.

Polyserositis is a frequent finding. Diffuse or localized fibrinous pericarditis is not specific, but there may be a more characteristic change, which varies from fibrinoid degeneration to a grossly thick gelatinous adherent pericardium. Similar involvement of the pleura may be seen, but more commonly there is only fibrinous pleuritis associated with the underlying pneumonia, a frequent complication. Perihepatitis and perisplenitis occur in about half the cases.[2]

The renal involvement produces striking urinary changes, described in some detail by Krupp.[10] In the same specimen, red blood cells, white blood cells, red-cell casts, fat bodies and fatty and waxy casts may be present. There are always abnormal amounts of protein. These changes, if they occurred in glomerulonephritis, would represent stages of the disease usually separated by years or decades. They would never all be found concurrently. The underlying renal lesion is most characteristically a peculiar alteration of the glomerular loops, which are irregularly thickened, rigid and deeply stained by eosin. This has been termed "wire looping" because of a fancied resemblance to bent wire.[2]

Diffuse changes in the blood vessels of all the organs are found. These consist of deposits of fibrinoid that progress to destruction of all the muscular and elastic elements.[2]

Most authors agree that the leukopenia is a characteristic and important feature of the disease, and secondary anemia is common and frequently severe.

One of the most consistent early clinical features of the disease is the arthritis, frequently mistaken for early rheumatoid arthritis or rheumatic fever. The subsequent involvement of the renal and cardiovascular systems and the polyserositis and the progressively downhill course are helpful in differentiating disseminated lupus from these clinical entities. This is well illustrated by the early difficulties in diagnosis in the 5 cases reported above.

Many authors[1-3] consider the predominant occurrence of disseminated lupus in the female one of its most striking characteristics. The incidence of 27 per cent of cases in the male at the Philadelphia General Hospital in the last decade is at variance with this concept, and is more consistent with the findings of other observers.[4, 5]

Summary

Of 36 cases of disseminated lupus erythematosus at the Philadelphia General Hospital in the years 1936 to 1949, 10 were in male patients, an incidence of 27 per cent. This is at variance with the generally accepted view that this disease is characteristically a disease of young adult women.

Five cases of disseminated lupus erythematosus in the male are presented to illustrate some of the clinical and pathological features of the disease.

References

1. Blount, S. G., Jr., and Barrett, J. T. Acute lupus erythematosus disseminatus: report of case in male with associated atypical verrucous endocarditis (Libman-Sachs). *Ann. Int. Med.* **23**:251-267, 1945.
2. Klemperer, P., Pollack, A. D., and Baehr, G. Pathology of disseminated lupus erythematosus. *Arch. Path.* **32**:569-631, 1941.
3. Baehr, G., and Pollack, A. D. Disseminated lupus erythematosus and diffuse scleroderma. *J. A. M. A.* **134**:1169-1174, 1947.
4. Rose, E., and Pillsbury, D. M. Acute disseminated lupus erythematosus — systemic disease. *Ann. Int. Med.* **12**:951-963, 1939.
5. Fox, R. A., and Rosahn, P. D. Lymph nodes in disseminated lupus erythematosus. *Am. J. Path.* **19**:73-99, 1943.
6. Rothman, S. and Felsher, Z. Subacute and acute disseminated lupus erythematodes. *M. Clin. North America* **31**:198-212, 1947.
7. Graffin, J. W., Taylor, C. B., and Hass, G. M. Clinical and pathological study of disseminated lupus erythematosus. *M. Clin. North America* **33**:79-94, 1949.
8. Montgomery, H. Pathology of lupus erythematosus. *J. Invest. Dermat.* **2**:343-359, 1939.
9. Libman, E., and Sacks, B. Hitherto undescribed form of valvular and mural endocarditis. *Arch. Int. Med.* **33**:701-737, 1924.
10. Krupp, M. A. Urinary sediment in visceral angiitis (periarteritis nodosa, lupus erythematosus, Libman-Sacks "disease"): quantitative studies. *Arch. Int. Med.* **71**:54-61, 1943.

SINGLE, CIRCUMSCRIBED, INTRATHORACIC DENSITIES*

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IN THE course of mass chest x-ray surveys for the detection of pulmonary tuberculosis, single, circumscribed densities are found that offer difficult problems in diagnosis and management. In the early years of the surveys, it was the usual procedure to follow these lesions by periodic examinations. Whenever an increase in the size of the lesion was noticed, the patient was referred for surgical treatment. All too often, the surgeon was then confronted with a malignant tumor that had become inoperable. Obviously, the opportunity for early removal had been missed. There were two reasons for the delay in the advice of operative intervention: unfamiliarity with the possibility of a silent, malignant chest tumor and the high mortality from intrathoracic operations. Remarkable advances in the technic and in the preoperative and postoperative care of thoracic patients have improved the results of thoracic operations. On the basis of the experience of the early years of mass surveys, we therefore decided to refer every patient with a single, circumscribed intrathoracic density for an exploratory operation as soon as the possibility of a malignant lesion could not be ruled out with a high degree of reliability. The general condition of the patient had to be good enough to permit major surgery since we soon learned that an exploratory operation usually led to a major surgical procedure — that is, to resection of a large portion of the lung or even of the whole lung.

In the past few years, we have seen 44 patients with single circumscribed intrathoracic densities, whom we tried to manage according to the stated plan. It is not our intention to present these cases in detail but to discuss some factors that prevented our plan from being as successful as we hoped it would be.

METHOD OF STUDY

With the co-operation of several local hospitals we used the following plan for the workup of patients with single, circumscribed intrathoracic densities. Whenever there was a family physician, the patient was referred to him with the following recommendations: history, including an inquiry about previous chest x-ray examinations (every attempt was made to obtain previous chest films); complete physical and fluoroscopic examinations; x-ray examination, including tomographic studies and special views as indicated; tuberculin test up to 1 mg. and skin tests for fungous diseases as indicated; sputum examination for tubercle bacilli and tumor cells and examination of gastric contents when necessary; bronchoscopy and angiocardiography on an ambulatory basis through the facilities of local hospitals whenever necessary; and search for possible primary tumors and x-ray examination of the gastrointestinal and genitourinary tracts at local hospitals.

In the majority of cases we were unable to establish criteria for the differentiation of a benign and a malignant intrathoracic lesion. Although we carried out many different examinations, as outlined above, only a few of them proved of real value for the differential diagnosis. The most significant procedure was tomographic examination. The demonstration of calcific deposits within an intrathoracic mass was considered a strong point in favor of a tuberculous lesion (tuberculoma§) or a benign lesion (hamartoma, chondroma or dermoid cyst). We did not feel justified in recommending the excision of these lesions as long as symptoms or change in size was absent. The coincidental development of a malignant lesion around an isolated calcified tuberculous process is very unusual and should not obscure the benignity of lesions with calcific deposits.

A positive coccidioidin or histoplasmin skin test was regarded as an indication for observation rather than exploration unless other pertinent findings were available. We did not recommend the routine removal of silent fungous granulomas since this question is still a controversial one. Examination of the sputum for tubercle bacilli and tumor cells was not of significant help at the stage in which we attempted to make a definite diagnosis.

EXPLORATORY OPERATION ADVISED AND ACCEPTED

Exploratory operation was advised after a complete workup and carried out on 21 patients. The operations were performed in various hospitals. The operative procedures varied from localized excision to pneumonectomy. There were 2 serious postoperative complications and 1 postoperative death. The diagnoses in the 21 cases are given in Table 1. The roentgenologic finding of a single, circumscribed, intrathoracic density can have a great variety of pathologic conditions as underlying causes. Seven of the 21 patients had a primary malignant lesion, 5 a bronchogenic carcinoma, 1 a pleural sarcoma, and 1 a thymoma. One patient had a single pulmonary metastasis, the primary tumor not being evident at the time of the complete medical examination. In 5 patients a tuberculoma was

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§The term tuberculoma is used in the roentgenologic sense to signify a round, dense lesion of at least 1 cm. in diameter. On pathological examination, it may represent a blocked cavity or a proliferative lesion.

resected. Calcifications could not be demonstrated roentgenologically in any of these cases. All 5 patients had an uneventful postoperative course. None

TABLE 1. *Diagnosis in 21 Cases of Accidentally Discovered, Single, Circumscribed, Intrathoracic Densities.*

DIAGNOSIS	NO. OF CASES
Primary carcinoma of lung	5
Metastatic carcinoma of lung	1
Fibrosarcoma of pleura	1
Thymoma	1
Neurofibroma of lung	1
Adenoma	1
Chondroma	1
Tuberculoma	5
Chronic lung abscess	2
Bronchiectatic cyst	1
Lipoid pneumonia	1
Pericardial cyst	1

of them developed a new tuberculous lesion during a follow-up period of at least a year.

The following case was typical:

CASE 1. A 65-year-old man had a survey chest film taken in December, 1949 (Fig. 1). The film was interpreted as showing a round density in the second left anterior intercostal space. A survey film taken two years prior was reviewed and found to be normal. Complete examination at the survey follow-up clinic revealed no additional findings. The patient was referred to his family physician. Bronchoscopy and sputum examination for tumor cells were negative. Nevertheless, the patient was explored and a bronchogenic carcinoma was found and removed.

FIGURE 1. *Roentgenogram of the Chest in Case 1.*
The density in the left-upper-lung field was discovered on routine examination. Operation was advised and accepted. (Diagnosis, bronchogenic carcinoma.)

EXPLORATORY OPERATION NOT ADVISED

In 13 patients we advised periodic observation of the lesion on the basis of the diagnostic examinations. In 8 patients calcific deposits were evident within the round mass. In 3 patients chest films taken several years previously were located. They showed identical findings with our survey films, and removal of the lesion was not advised for this reason. Two patients with a history of residency in the Southwest United States had positive coccidioidin tests. We thought we were dealing with fungous granulomas and did not advise exploratory operation. The lesions in all but 1 of the 13 patients for whom observation was advised showed no change over a follow-up period of at least two years. The exception was a round mass with calcific deposits that increased in size after a period of stability of a year. Operation was then advised but refused. After this concentric growth the lesion again remained stable for eighteen months. Our tentative diagnosis in this case was hamartoma.

FIGURE 2. *Roentgenogram of the Chest in Case 2.*
The density in the left-upper-lung field contains calcific deposits. There was no change during twelve years of observation. (Probable diagnosis, tuberculoma.)

The following case is typical of those in this group:

CASE 2. A 49-year-old man had a survey chest film taken on March 17, 1947, that showed a round density with calcifications in the left-upper-lung field. The patient was

asymptomatic. A survey film taken 9 years previously was located. It showed an identical lesion. The patient had been followed for another 3 years without showing any change in the chest film. The most likely diagnosis is tuberculoma of the left upper lobe (Fig. 2).

Exploratory Operation Advised but Not Carried Out

In addition to the 21 patients operated on there were 10 patients in whom a malignant lesion could not be ruled out and exploratory operation was advised. On these 10 patients the operation was not carried out for various reasons. Three patients rejected the possibility of having a serious disease because they had no symptoms. They did not even consult their family physicians as suggested by the clinic physician. Six patients sought the advice of their family physicians, who advised watchful waiting, mainly because the patients were asymptomatic. One patient was hospitalized. After a negative bronchoscopic examination, she was referred to the Out Patient Department for observation, although the possibility of a malignant lesion was entertained at the time and later on definitely established. We have been able to follow all 10 patients for a minimum period of one year. One of the 10 patients died, the clinical diagnosis being primary pulmonary cancer. Four patients offer the clinical picture of advanced cancer (metastasis, continuous growth of the original tumor). In 3 patients the lesions have not changed during a follow-up period of twelve, twenty-four and thirty months respectively. In 2 patients, the lesions increased in size after a period of stability of approximately eighteen months, only to remain stationary again for the remainder of the observation period (eighteen months).

The following case is an example of those in this group:

Case 3. A 58-year-old man was admitted to the survey follow-up clinic on October 20, 1947. He had been treated for syphilis (contracted in 1923) in 1937, 1938 and 1941. The blood test had remained positive in spite of several courses of arsenic and bismuth treatment. He had had pleurisy in childhood and a productive cough for many years. In 1943 he had had blood-streaked sputum. A chest x-ray film taken at that time was reviewed and found to be normal. Physical examination was negative. A chest film (Fig. 3) in October, 1947, showed a large, circumscribed density in the left-upper-lung field. A neoplastic lesion was considered the most likely diagnosis, and the patient's physician was advised to this effect. The patient was treated with penicillin on the assumption that the pulmonary lesion represented a gumma. On the basis of fluoroscopy, the family physician believed that the lesion had first decreased slightly but later enlarged. When the patient had a small hemoptysis in April, 1948, 6 months after the initial examination, he was hospitalized. An exploratory operation revealed an inoperable bronchogenic carcinoma.

Discussion

As a by-product of mass surveys for the detection of pulmonary tuberculosis, a number of silent chest masses were discovered among which there were malignant tumors frequently undistinguishable without exploratory operation from benign tumors and inflammatory, cystic or granulomatous lesions. To avoid loss of valuable time, it was recommended that every patient in whom a malignant lesion could not be ruled out after a complete examination be explored. Exploratory operation was rejected, however, by 10 out of 31 patients in whom it was advised. Six patients refused to accept a surgical procedure on the advice of their physicians. The family physicians of these six patients based their

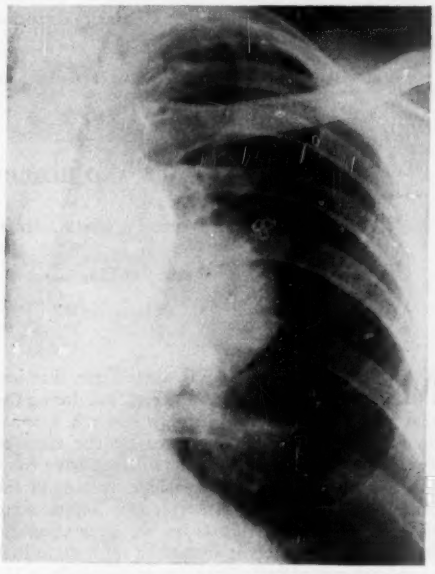

Figure 3. *Roentgenogram of the Chest in Case 3. The density in the left-upper-lung field was found on routine examination in October, 1947. Operation was advised but not accepted at that time. Exploratory operation in April, 1948, after hemoptysis revealed an inoperable bronchogenic carcinoma.*

recommendation mainly on the absence of symptoms. It is very important to embark on an extensive educational program to increase the general practitioner's knowledge of the asymptomatic intrathoracic tumor and to stress the value of early exploration of an asymptomatic, undiagnosed, isolated chest mass.

We are aware that no information is given concerning the total number of survey x-ray films among which the 44 cases of circumscribed lesions were found, nor did we state the age and sex distribution. The reason is that no adequate data are available on the prevalence of malignant tumors in age-specific survey groups. Some information has been obtained from large city-wide surveys carried out

by the United States Public Health Service* but detailed reports of prevalence and incidence of malignant tumors in mass chest x-ray surveys are necessary before the significance of this method for case finding of malignant tumors can be evaluated. A study to this effect is being carried out by this department, and the result will be published in a future report.

Summary

In the course of mass chest x-ray surveys for the discovery of pulmonary tuberculosis, 44 patients with single, circumscribed intrathoracic densities were seen.

In 31 patients, a malignant lesion could not be ruled out after a complete examination, and early exploratory thoracotomy was advised. Twenty-one patients underwent exploratory thoracotomy. Seven primary malignant lesions and 1 metastatic lesion were removed. Ten patients refused the exploratory operation, 6 of them on the advice of family physicians. Five of the 10 patients were subsequently shown to have a definitely malignant lesion.

Thirteen patients were observed by periodic examinations, since the initial workup suggested a benign lesion. None of the 13 patients gave evidence of a malignant lesion during a follow-up period of at least two years.

The early exploration of every single intrathoracic mass is recommended whenever a malignant lesion cannot be reliably ruled out.

*Overholt, R. H., and Schmidt, I. C. Silent phase of cancer of lung. *J. A. M. A.* 141:817-821, 1949.

THE INCIDENCE AND HEREDITY OF MUSCULAR DYSTROPHY

A Study of Seventy-One Patients Admitted to the Massachusetts General Hospital

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MUSCULAR dystrophy is a condition that has aroused little popular interest, but during the past year a muscular dystrophy research foundation has been granted a charter by the state of Texas, and from the University of Utah have come two papers based on the genealogic records of the Church of Jesus Christ of Latter Day Saints, identifying affected individuals in six generations.[1, 2] In view of these facts it seemed of interest to look at the records of one of the oldest hospitals in the United States.

The file by diagnosis in the Record Room of the Massachusetts General Hospital goes back eighty years, to 1870, and does not include patients seen in the Out Patient Department only. The first patient on whom a diagnosis of muscular dystrophy was made entered the hospital in 1904, and the next in 1909, but previous to these dates, under the heading "progressive muscular atrophy," were 5 patients with what is today known as muscular dystrophy. The first patient entered the hospital in 1870, and in the volume containing his record there is his picture drawn by Edward Waldo Emerson (Fig. 1) first-year student at Harvard Medical School in 1870 and house pupil, West Surgical Service, Massachusetts General Hospital in 1874 (son of Ralph Waldo Emerson and instructor at the Boston Museum of Fine Arts in art anatomy after 1883). This shows the characteristic scapulohumeral type of muscular atrophy. These early patients had had neurologic examinations and electric tests performed by Dr. James Jackson Putnam, house pupil (intern) in 1870, appointed electrician to the Massachusetts General Hospital in 1874, indicating that he was the first neurologist to the hospital (the professorship of neurology at Harvard Medical School bears his name). After making a study of the records from 1870 through 1947 I believe there were 71 patients who had progressive muscular dystrophy. Thirty-five of these entered the hospital during the last eleven years covered by this study. From 1870 to 1916 only 6 patients were mentioned. In addition, there were 9 patients with two other characteristic forms of hereditary muscular atrophy: 3 had myotonia atrophica and 6 had peroneal muscular atrophy (Charcot-Marie-Tooth).

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Race

Since muscular dystrophy is hereditary, note was made of the nationality and birthplace of patient and parents. Of the 71 persons with progressive muscular dystrophy 28 were Americans of British stock, 8 were Irish Americans, 11 were Italians, 9 were French Canadians, 7 were British Canadians, 7 were Polish, 3 were Jewish, 2 were Greek, 1 each were Irish, Lithuanian and Norwegian. There were 2 of mixed parentage — Italian and German, Italian and French Canadian. The increase in number of patients with muscular dystrophy during the last twenty years of the study was due to the foreign-born population. The total of those

with native-born New England parents was 15. All but 1 of the 6 early patients were of Yankee stock; from 1916 to 1937 and from 1937 to 1947 there were 5 with parents born in New England.

Characteristic Types

There can be no rigid classification of progressive muscular dystrophy, and the many types and proper names used in textbooks have done little except mystify the medical student and the physician. In this series the cases of progressive muscular dystrophy fell into 3 types: those with pseudohypertrophic muscles, those with a generalized muscular atrophy starting in the thighs, and those with a generalized muscular atrophy starting in the scapulohumeral musculature.

Of the patients with the first type 26 had their first symptoms in the first decade of life, and 2 of these were girls. There were 8 of the older group who had hypertrophy of calves, and 1 woman had hypertrophy of shoulders and upper arms. Five of the 8 were women, and the onset of muscular weakness came in the second, third, fourth and fifth decades of life.

The second type, a group of 21 with symptoms starting in the legs, had atrophy without hypertrophy or facial involvement. There were 9 in the second, 7 in the third, 3 in the fourth, 1 in the fifth, and 1 in the sixth decade. Eight were females, and 13 were males. Three women and 1 man had atrophy and weakness of the thighs only; 1 woman had involvement of thighs and trunk only.

Of the 15 patients in the facioscapulohumeral group, 8 were females, and 7 males. Three had had first symptoms in the first, 7 in the second, 4 in the third, and 1 in the fourth decade. It is of interest that the 3 children under ten were all girls. Scapulohumeral atrophy is much more common than facioscapulohumeral, and in this series facial atrophy was mentioned in connection with only 2 patients. If the atrophy began in scapulohumeral muscles, the patients were classified in this group, though later the trunk and leg muscles were involved.

Heredity

Fourteen patients had siblings with progressive muscular dystrophy, and these were almost all in the childhood or adolescent groups. There were 2 pairs of sisters in middle life, 3 of whom had been in the hospital, and 3 had the typical picture of hypertrophy of the calves. Two of these I have examined and also a brother with a mild but similar form of the disease. Only 1 afflicted member of the family in the preceding generation was mentioned in all 71 records. That was in the family history of the sisters mentioned above; their father's half sister had had the same difficulty in walking that the patients had. At first thought the lack of affected members in preceding generations is laid to poor histories and poor memories on the part of the patient and his family. However, such a marked deformity of the locomotor system in a relative is not easily forgotten and certainly not in a parent or child. The family history was always taken in detail on these records, for the physician was dealing with a known hereditary disease. Recessive genes must be present in and received from both parents, and since the patient receives only half his genes from each parent, many generations in succession may be free of overt manifestation. Therefore, in dealing with recessive genes in human beings, one must rely for the most part on sibling inheritance, and even there 20 per cent probably is an average figure.

Figure 1. *Drawing of First Patient on Whom a Diagnosis of Muscular Dystrophy Was Made.*

Other Hereditary Defects

Of the 71 patients with progressive muscular dystrophy 5 had pes cavus, and 2 pes equinovarus, 3 were mentally deficient, and 3 had progressive nerve deafness.

Pathology

Many of the later patients had had muscle biopsies, but there was 1 unusual patient who was studied at autopsy and was discussed in the weekly clinicopathological conference at the hospital.[3]

He was a young man of twenty-four who had had muscular dystrophy since eight years of age. He died with cardiac hypertrophy and dilatation, mural thrombi of the left ventricle and renal and splenic infarcts. The myocardium showed the same changes found in dystrophic skeletal muscles — that is fibrosis and infiltration of fat between the muscle fibers. During the discussion Dr. Walter Bauer mentioned that he and Dr. Joseph Aub had given adrenocortical extract without benefit to a number of patients with muscular dystrophy.

Treatment

As early as 1929 there were notations in the records that adrenocortical extract had been given patients. Thyroid, pituitary extract and testosterone had also been used, and none had given beneficial effect. In 1950 ACTH was given a child, and a temporary increase in muscular weakness resulted.

Discussion

The geneticists do not agree on either the clinical types or the method of inheritance in progressive muscular dystrophy. Dr. Julia Bell,[4] on the basis of 1228 cases from the literature and 113 records from Queen's Square Hospital in London, stated that there are three clinical groups: pseudohypertrophic, atrophic without facial atrophy, and those with facial atrophy. Her three genetic groups are dominant, recessive and sex-linked recessive, suggesting that the same main gene can produce the three clinical groups. Dr. F. H. Tyler and Dr. F. E. Stephens, a geneticist of Salt Lake City, Utah, on the basis of 175 patients examined, believe that there are two clinical types, pseudohypertrophic of childhood in the male and facioscapulohumeral of early adolescence affecting both sexes, and 2 genetic types, sex-linked recessive and mendelian dominant.[1,2] They had two great advantages over Dr. Bell: the actual examination of the patients, and the genealogic records of the Church of Jesus Christ of Latter Day Saints. On the other hand, their facioscapulohumeral dystrophies were limited to one "kindred," and it is usual for the same clinical type to be repeated in the same family. This "kindred" consists of 58 living patients and the genealogic records of 1250 persons in six generations, of whom 159 were or are affected. The ingenious charts showing the dominant descent of facioscapulohumeral dystrophy from parent to child are published but not those relating to the recessive childhood pseudohypertrophic type. It seems to me that the sex-linked recessive type of inheritance will be difficult to prove. The patients do not reproduce, and the data on previous generation seem to yield little. Perhaps the Utah material will be more helpful.

On the basis of the 71 patients admitted to the Massachusetts General Hospital it appears that the earlier the onset of dystrophy, the more severe the disease, and that the male is more prone to this early onset but the female is by no means immune. Many patients had weakness and atrophy starting in the legs and affecting later the trunk muscles and the upper extremities. Dr. Bell's second class of atrophy without hypertrophy or facial involvement seems to include this group, which in this series was larger than the scapulohumeral group.

To get a better idea of the frequency of appearance of muscular dystrophy in Boston and in New England the record librarians of the Boston City Hospital and the Children's Medical Center were consulted. From 1931 to 1944 at the Boston City Hospital the discharge diagnosis of muscular dystrophy had been made in 37 cases. From 1902 to 1942 at the Children's Medical Center 55 patients had received the diagnosis of muscular atrophy, and from 1937 to 1949, 28 that of muscular dystrophy. Although there was a total of 192 diagnoses in the three hospitals, many patients had been in two of the institutions.

Summary and Conclusions

In New England, hereditary muscular dystrophy is not partial to any one nationality of the white race.

Of those with native-born parents only 15 had parents born in New England.

Of the 71 patients 35 had pseudohypertrophic muscular dystrophy. Of the two atrophic types the onset had been in the muscles of the thighs in 21 and in the facioscapulohumeral group in 15. In all, there were 47 males and 24 females.

Fourteen patients had siblings with muscular dystrophy, but none had a parent or child with muscular dystrophy.

Other hereditary defects found were pes cavus (5 patients), pes equinovarus (2 patients), feeblemindedness (3 patients) and progressive nerve deafness (3 patients).

Though the diagnosis of muscular dystrophy has not occurred frequently in the early records of the Massachusetts General Hospital, the last two decades have seen a marked increase. There was no evidence of mendelian dominant inheritance among the patients with facioscapulohumeral atrophy similar to that in the large Utah "kindred."

The inheritance of the three types of muscular dystrophy in this series was recessive.

I am indebted to the staff of the Record Room, Massachusetts General Hospital, for their co-operation in making available the records on which this paper is based.

References

1. Tyler, F. H., and Wintrobe, M. M. Studies in disorders of muscle. I. Problem of progressive muscular dystrophy. *Ann. Int. Med.* **32**: 72-79, 1950.
2. Tyler, F. H., and Stephens, F. E. Studies in disorders of muscle. II. Clinical manifestations and inheritance of facioscapulohumeral dystrophy in large family. *Ann. Int. Med.* **32**:640-660, 1950.
3. Case Records of the Massachusetts General Hospital (Case 33311). *New Eng. J. Med.* **237**:163-167, 1947.
4. Bell, J. *On Pseudohypertrophic and Allied Types of Progressive Muscular Dystrophy.* Vol. 4. Part 4. In *The Treasury of Human Inheritance.* Edited by R. A. Fisher. London: Cambridge University Press, 1942.

FIFTY YEARS OF MEDICAL PROGRESS

MEDICINE AS A SCIENCE: OBSTETRICS

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THE obstetrician occupies an unusual place in the practice of medicine. Most of his patients are not sick, but are performing the natural function of reproduction. If this function were always normal his cares would be few indeed, but complications and accidents may occur suddenly and with disquieting frequency. He must also be a surgeon, an internist and, on occasions, a pediatrician. His work demands the most meticulous aseptic technic, his patients may suffer from any medical ailment, and he has the first opportunity to detect disease or abnormality in the newborn infant. These are the factors that make his specialty one of the broadest and most interesting in medicine.

The ideal of good obstetrics is, whenever possible, to bring every gravid woman through her pregnancy, confinement and puerperium alive and well and with a living, healthy infant. How nearly this goal is approached today and how much improvement there has been in the last fifty years is a matter of official record. According to the National Office of Vital Statistics[1] the maternal mortality for the birth registration area in 1915, the first year for which the number of births is available, was 6.1 per 1000 live births. In 1947, the last year with published results, the rate was 1.3. This is the lowest national rate for the United States, and it is also the lowest rate for any country, large or small.

Massachusetts has recorded its vital statistics since 1842. During 1900 there were in this state 70,513 live births and 278 maternal deaths, a mortality rate of 3.9.[2] In 1948, the last year with a published report, there were 100,434 live births and 74 maternal deaths, a rate of 0.7.[3] If the rate of 1900 had been current in 1948, there would have been 392 maternal deaths instead of 74 — that is, 318 more mothers would have lost their lives.

These statistics show that in the last thirty-two years deaths in childbirth throughout the nation have been reduced about four and three-fourths times and that in the last fifty years in Massachusetts they have been reduced about five and a half times. This achievement is not fortuitous but has been brought about by the efforts of the physicians who practice obstetrics. It is doubtful if any other branch of medicine can produce so good a record.

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Stillbirth is a definite obstetric problem. Stillbirth rates are available from both the National Office of Vital Statistics and the Commonwealth of Massachusetts. The first figures from the federal source[1] are for 1922, when the rate was 39.2 per 1000 live births; whereas in 1947, the last year reported, it was 23.7, a reduction of 40 per cent. The stillbirth rate in Massachusetts[2] for 1900 was 40.7 per 1000 live births; but in 1948,[3] the last year published, it was only 19.3, a reduction of over 50 per cent.

Neonatal death rates for so long a period as fifty years have not been reported by either the federal Government or Massachusetts. Statistics from the Boston Lying-in Hospital,[4] however, show the improvement made at that institution. In the decade 1893–1902 the neonatal death rate was 25 per 1000 births, live and still, but in 1949 it was 10, or 60 per cent, less. The premature death rate for the same decade was 10 and in 1949, 7, or 30 per cent, less. Even more satisfactory was the improved neonatal death rate of infants of over 5½ pounds. In 1893–1902 it was 15, and in 1949 it was 5, or 66.6 per cent, lower.

The chief causes of maternal deaths have always been puerperal infection, hemorrhage and eclampsia, and it is mainly by a reduction in the deaths from these sources in the last half-century that the mortality in childbirth has been lowered in so striking a manner.

PUERPERAL INFECTION

Let us look back fifty years to the conditions prevailing at that time. Obstetricians had been aware of the contagious nature of childbed fever since the days of Holmes and Semmelweis in the middle of the nineteenth century and had recognized its bacterial origin since Pasteur demonstrated the role of the streptococcus in 1879. The aseptic technic of the best maternity clinics then was quite the equal of that employed by the leading surgical authorities. Nevertheless, a number of patients continued to die of puerperal infection. Bacon,[5] of Chicago, reported that in 1895 7.3 per cent of the women between twelve and fifty dying in that city succumbed to childbed fever, and Reynolds,[6] of Boston, said that during 1893 he had seen in consultation 28 patients who were victims of the disease and that 7 had died. In 1910 Williams[7] (Fig. 1) stated that although in maternity hospitals the

death rate was only a fraction of 1 per cent, it was much higher in home deliveries conducted by private physicians. On the other hand, sporadic epidemics occurred from time to time even in the most carefully conducted maternity clinics.

Bacteriologic investigations have shown that hospital infection rarely takes place in the delivery rooms, but that it is most often literally post par-

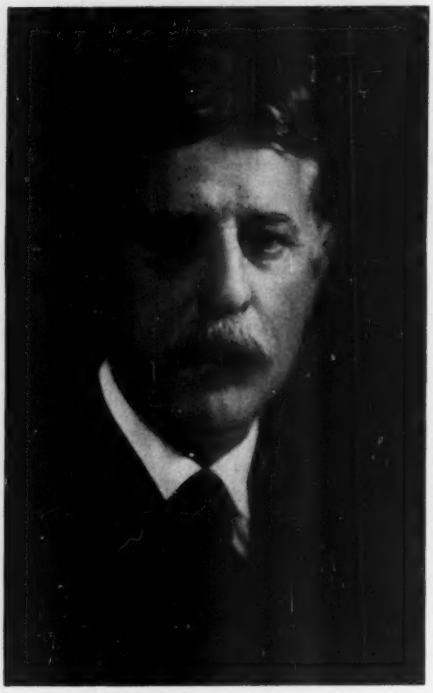

FIGURE 1. *John Whitridge Williams (1866–1931), the Most Distinguished Obstetrician of the Early Twentieth Century. He made obstetrics a science as well as an art.*

tum in that it is passed from patient to patient while they are convalescing. The chief mediums of inoculation are the hands of the nurses and the utensils used in attending to the hygienic needs of the patients. The abolition of manual methods in conducting the perineal toilet and the substitution of individual sterile instruments, the prompt isolation of infected patients, the requirement that all personnel in contact with puerperal women wear masks and head coverings and have throat cultures negative for streptococci and the prohibition of all visitors with respiratory infections have effectually eliminated hospital epidemics.

From 1900 to 1936, when Colebrook[8] introduced sulfanilamide into obstetric practice, the death rate from puerperal infection at the Boston Lying-in Hospital was 1 in 774 patients delivered in all departments of the institution. From the advent of the sulfonamides to the clinical introduction of penicillin[9] in 1944 the death rate from the same cause fell to 1 in 1595 deliveries. In the six years from 1944 to 1950 there was only 1 death from puerperal infection in 32,667 confinements.[10]

Two other factors have contributed greatly during this period in lessening the frequency of puerperal infection. One is the abandonment of such shock-producing operations as manual dilatation of the cervix and high forceps delivery. The other is the substitution, whenever possible, of rectal for vaginal examinations.

In modern obstetric clinics puerperal infection, except under the most unusual circumstances, belongs to the past. The lives of thousands of mothers, which not so long ago would have been lost, are now being preserved. The younger directors of maternity clinics today are indeed fortunate, for they are spared the sense of failure, frustration and anguish that on the occasion of a hospital epidemic so appalled their predecessors.

Cesarean section was closely involved with puerperal infection because so many deaths that followed it were from peritonitis. At the beginning of the century it had only been eighteen years since Sänger[11] had made the operation comparatively safe by the simple expedient of suturing the uterus, but it was already being performed with greater confidence. In carefully selected cases, however, the mortality was as high as 5 per cent, and it was generally admitted that it was too dangerous for patients who were actually or even potentially infected.

In the early part of the century practically all cesarean sections performed in this country were of the classic type, but in 1912 Krönig,[12] seeking a safer method for the potentially infected patient, devised the first of the transperitoneal lower-segment operations. He reflected the vesical flap of peritoneum, displaced the bladder downward and incised the lower portion of the uterus in a vertical direction. This operation is probably in most general use today, and is the one to which DeLee gave the high-sounding title of laparotrachelotomy. A similar procedure, but with a crescentic transverse incision in the lower segment, was devised by Kerr[13] in 1921. This technic is also used by many, and there is little choice between the two.

As early as 1909 Latzko,[14] believing that a strictly extraperitoneal approach was necessary to ensure safety in the infected or even the potentially infected patient, described the first practical, truly exterior cesarean section. He exposed the lower uterine segment by displacing the bladder to one side. Two American obstetricians, Waters[15] and Ricci,[16] have based their operations on the work of

Sellheim,[17] who advocated the retrovesical approach. These two types of operations are most generally used at present when an extraperitoneal section is indicated.

Before the perfection of the extraperitoneal operation when an obstetrician was faced with an infected patient who could only be delivered by cesarean section his only recourse was to follow the operation by a hysterectomy. Collected statistics[18] show that under these circumstances extraperitoneal cesarean section is two or three times safer.

Now that the effectiveness of the sulfonamides and the antibiotics has been established, some operators, when dealing with potentially or actually infected patients, have relied on the drugs to overcome the risk attending the lower-segment operation. Such patients, however, are entitled to every resource of science, which includes not only the latest therapy but the extraperitoneal operation as well. Neither the sulfonamides nor the antibiotics are infallible, but there is a disquieting tendency in all branches of surgery to rely on them to cover inadequate technic.

When performed on uninfected patients either the classic or the lower-segment operation now has a high degree of safety, and from the point of view of mortality alone there is little difference between them. Like members of the two political parties on the Island of Lilliput, one may open the egg either at the large or at the small end. In general, however, the lower-segment operation is followed by a smoother convalescence, and there is distinctly less danger of rupture of the uterus in a subsequent pregnancy.

The mortality of cesarean section in hospitals that accept emergency cases as well as service and private patients is now about half of 1 per cent.[19] Many times the operation is lifesaving for mother or child, and to the well trained and intelligent obstetrician it offers a solution for many difficulties.

Hemorrhage

Transfusion has been the key to the problem of hemorrhage in obstetrics, and in this connection three dates are important: 1907, when Jansky[20] classified all human bloods into the four main groups; 1914, when Lewisohn[21] devised the simple technic of citrate transfusion; and 1940, when Landsteiner and Weiner[22] demonstrated the Rh factor and its bearing not only on transfusion but also on the production of erythroblastosis fetalis.

The chief ante-partum causes of excessive bleeding are abortion and miscarriage, extrauterine pregnancy, placenta praevia and premature separation of the normally implanted placenta. The hemorrhage from abortion and miscarriage is seldom sufficient to endanger life and since their chief interest lies in their pathological aspect and in modern methods for their prevention, they are considered later.

In 1883 Lawson Tait[23] established the operative treatment for *extrauterine pregnancy*. Up to that time the only surgical interventions had been in cases in which the fetus had died and had become encapsulated; but by the beginning of the twentieth century, owing to the investigations of Schauta,[24] who in 1891 reported that the results were fifteen times better by operation than with expectant treatment, extrauterine pregnancy came to be recognized as an emergency and to be treated as such. As transfusion became common practice the death rate fell so that today it is from 1 to 1.5 per cent.

In the treatment of *placenta praevia* cesarean section now occupies the most important place. It avoids laceration of the friable cervix, which is often caused by rapid methods of pelvic delivery, and it affords the living infant the best chance of survival — a fact that Dudley[25] noted as long ago as 1900.

At the beginning of the century Bürger and Graf[26] reported a maternal mortality in placenta praevia of about 5 per cent and stated that only half the infants survived. At present the risk to the mother is approximately 1 per cent, but the fetal mortality — about 35 per cent — remains high. But if nonviable infants, very premature infants and those dead *in utero* when the patient is first seen are excluded the net fetal loss is about 11 per cent.[27]

Two factors have contributed to the salvage of maternal life in this condition: transfusion, as in obstetric hemorrhage from whatever cause, and increased resort to cesarean section. Cesarean section gives the infants a distinct advantage. Findley[28] proved this conclusively in 1938, when he collected 47,838 cases from the literature and found the fetal mortality with pelvic delivery to be twice that with cesarean section.

The modern diagnosis of placenta praevia is greatly aided by the soft-tissue x-ray film, described by Snow and Powell[29] in 1934, and by the cystogram, introduced by Ude, Weum and Urner[30] in the same year. Information so obtained is not always conclusive, so it is confirmed in the operating room under full aseptic precautions with assistants ready and all preparations made for cesarean section or for the induction of labor by rupture of the membranes, the introduction of a dilating bag or the performance of a Braxton-Hicks version. These precautions avoid the danger of a serious hemorrhage, which sometimes results even from a simple vaginal examination.

With a patient who has bled only slightly and whose living infant might benefit by several weeks more of intra-uterine life, many obstetricians believe that it would be justifiable to put her to bed in a hospital under constant observation, provided there is a competent resident staff who can operate promptly if an alarming hemorrhage occurs. To

send a woman home who is known to have placenta praevia, however, is to court disaster.

In 1901 Holmes[31] collected 200 cases of the serious type of *premature separation of the normally implanted placenta* and showed that almost a third of the patients and seven eighths of their babies died. Eleven years later Couvelaire[32] published his classic description of the mottled appearance of the uterus found in internal concealed hemorrhage and the dissociation of its musculature caused by edema and the effusion of blood. Albuminuria, which accompanies over a third of the cases, had been noted by Winter[33] in 1885. Soon it was recognized that a fatal outcome more often followed retention of blood within the uterus than the existence of toxemia or nephritis.

At the beginning of the century most patients with this condition were delivered — as were those with placenta praevia — by *accouchement forcé*, and with even worse results. Although such women are suffering from hemorrhage and shock and often have anuria, many obstetricians then turned to cesarean section, supported by transfusion, as a solution of the difficulty. In 1931 conservative treatment, based on the work of Fitzgibbon,[34] of Dublin, was introduced by Polak[35] into this country: this consists of rupture of the membranes, the application of a tight abdominal binder, and small doses of pituitary extract. In selected cases it has a mortality of 3 per cent. The death rate in cesarean section when performed routinely for this condition is about 15 per cent, and when it is followed by hysterectomy it is usually twice as high. Rational therapy indicates a trial of conservative measures, followed by a resort to cesarean section if the patient fails to make progress. For the otherwise uncontrollable oozing that sometimes follows delivery the injection of fibrinogen, as advocated by Moloney, Egan and Gorman[36] in 1949, has much to offer.

The fetal mortality is naturally very high. In about two thirds of the cases the infants are dead when their mothers are admitted to the hospital or are so premature that survival is rare. The fetal death rate shows no improvement over that of fifty years ago, nor, because of the sudden nature of the attacks, is there likely to be any change.

The treatment of the external type of hemorrhage is a much simpler problem. Delivery through the pelvis after rupture of the membranes, if necessary, or by cesarean section if the bleeding is alarming results in a low maternal mortality, but about two thirds of the infants are lost.

Little information can be found in the literature of fifty years ago about *post-partum hemorrhage*. Data from the Boston Lying-in Hospital,[37] however, show that the death rate from this cause from 1909 to 1918 inclusive was 1.2 per 1000 deliveries, whereas in the five years ending in 1948 it was 0.1. Obviously, transfusion has played the major part in this reduction, but credit must be shared with pituitary extract, introduced as an oxytocic by Blair Bell[38] in 1909, and with the new and more effective preparations of ergot. In 1928 Kamm and his associates[39] isolated oxytocin, the principle in pituitary extract that acts primarily on the uterine musculature, and in 1932 Moir[40] separated from ergot the active principle ergonovine. Of prime importance also is the present policy followed by conservative obstetricians of delaying the completion of the third stage of labor until all the signs of placental separation have appeared.

Eclampsia

In the last half of the nineteenth century most obstetricians enjoyed a comfortable state of mind about the cause of eclampsia. As long before as 1843 Lever[41] had found albumin in the urine of women with puerperal convulsions; moreover, edema and stupor usually preceded or accompanied the fits. The sphygmomanometer, invented by Riva Rocci in 1896 and used by the more progressive, showed that such patients had high blood pressures; the picture was one of uremia. But an element of doubt had been raised by Schmorl[42] in 1893 and by Lubarsch[43] in 1896. These observers had found changes in the livers of women dying of eclampsia that never appeared in nephritis. Faced with this information at the beginning of the twentieth century obstetricians abandoned the concept of uremia and assumed that the disease must be caused by some unknown toxin.

Owing to the independent work of Acosta-Sison[44] and of Davidson,[45] both published in 1931, it is now known that these characteristic lesions in the liver may consist of areas of hemorrhage, necrosis or fatty degeneration and that they may be found at the central, midzonal or peripheral portions of the lobules. In 1924 Fahr,[46] while studying the kidney, found that the glomeruli were increased in size because of swelling of their capillary walls and that there was a relative absence of blood cells in the loops, which created the picture of local ischemia. Bell,[47] using the azo-carmine stain, in 1932 demonstrated thickening of the capillary basement membrane. It is believed that these changes are due to thickening and partial occlusion of the afferent arterioles. Nevermann[48] and Hinselmann[49] noted spasm of the capillaries in the nail beds of patients with eclampsia, and Mylius[50] found similar changes in the retinas. From this evidence most obstetricians now accept the hypothesis that eclampsia is a disease characterized by spasm of the small arterioles and that its signs and symptoms are produced by this phenomenon.

The actual cause of eclampsia remains unknown. During the half century just ended between twenty and thirty theories have been offered in explanation, but none have merited general acceptance. The most recent hypothesis — that of Smith and

Smith[51, 52] — possesses novelty and plausibility. They note that in normal pregnancy there are high and balanced urinary levels of estrogens and progesterone at the thirty-eighth week and a marked decrease preceding spontaneous labor. This high level indicates a minimal destruction of estrogens and, hence a diminished amount of oxidation products which stimulate the syncytium. This leads to degeneration of the syncytium and therefore to a diminution in the amount of estrogens elaborated by it. It also causes the onset of labor by withdrawal of hormonal support to a uterus stretched to capacity. Since the same train of circumstances precede the appearance of pre-eclampsia, the Smiths postulate that an injurious protein resulting from syncytial degeneration is absorbed into the blood stream and causes toxemia. The histologic studies of Tenney and Parker[53] bear out this hypothesis.

Following advances in chemical laboratory methods, a vast amount of effort has been expended on the study of eclampsia. On the whole, the results have been meager. It has been found that the plasma proteins, normally low in pregnancy, may be even more reduced in toxemia, and that there is a tendency toward a reversal of the albumin-globulin ratio. Sodium, which is retained in normal pregnancy, is accumulated to an even higher degree in this condition.

Eclampsia is much less of a menace than it was fifty years ago, for it occurs much less often and the mortality is lower. In 1897 Reynolds[54] estimated that it appeared once in every 400 labors; in the last six years at the Boston Lying-in Hospital there was only 1 case in every 1636 labors.[55] The greatest factor in this remarkable reduction has been the development of ante-partum care, which did not exist in maternity clinics until the second decade of this century. Before then patients merely registered at the hospital and reported when they started in labor. There is no reason to believe that conditions were better in private practice. Such patients might prove later to have eclampsia, placenta praevia, contracted pelvis or any of the complications of pregnancy or labor. Routine urinalysis and blood-pressure determinations, with the prompt admission of pre-eclamptic patients for hospital care and termination of pregnancy when there was no improvement, have been the most effective measures in preventing the onset of eclamptic convulsions.

As in the other serious complications of pregnancy the treatment of eclampsia has improved greatly in the last fifty years. At the beginning of the century, when *accouchement forcé* was in vogue, the death rate was about 30 per cent, and many women succumbed to the treatment as well as to the disease. Cesarean section, with a mortality of 20 per cent, offered little improvement.

About twenty-five years ago Stroganoff[56] advocated the conservative treatment of eclampsia with morphine, chloral and chloroform, and postponed the termination of pregnancy until the patient's condition had improved. Although his method in detail has generally been abandoned, the concept of conservatism now governs the treatment of this disease. At the same time Lazard and his associates[57] introduced the use of magnesium sulfate to control convulsions and lower the blood pressure. This drug, now given intramuscularly, is at present the chief therapeutic agent. Employing it and adjuvant measures, Stander[58] reported a death rate of 1.5 per cent. In 1940 Bryant[59] revived the use of *Veratrum viride*, which, combined with magnesium sulfate, he reported as giving a mortality rate of 1.6 per cent. The most important element in modern treatment, however, is postponement of all measures to effect delivery until the patient's poor condition is stabilized.

In 1907 Edgar[60] stated that the fetal mortality in eclampsia was 50 per cent; today it is about 30 per cent. Because the onset of convulsions greatly jeopardizes the infant, the termination of pregnancy before this stage is reached increases the fetal salvage to a marked degree.

Pernicious Vomiting

Fifty years ago Williams[61] classified excessive vomiting in pregnancy as toxic, reflex or neurotic. There is no evidence that pernicious vomiting is due to a toxemia. Among women who vomit excessively in early pregnancy no more will develop pre-eclampsia or eclampsia than those who do not vomit at all. No reflex mechanism has been demonstrated. There is little doubt, however, that a definite neurotic element is often involved, and therapeutic measures with this fact in mind are often effective.

As knowledge of the endocrines developed it was natural that they should be called upon to explain this condition, but so far no convincing evidence has been presented. It is unlikely that deficiency of the corpus luteum hormone is at fault, for the abortion rate is only a seventh of the normal expectancy.[62] Berkwitz and Lufkin,[63] in 1932, called attention to the polyneuritis that may occur in advanced stages of pernicious vomiting. Some have believed it due to deficiency in vitamin B_1, but large doses of thiamine alone have failed to cure it and additional measures must be used.

In severe cases excellent results follow tube feeding and the employment of fluids intravenously. The principle is the treatment of starvation, and is based on the fact that the fatty degeneration found in the liver and kidneys differs in no way from what is encountered in that condition. Therapeutic abortion is rarely needed, and deaths are so uncommon that many clinics report series of 250 or more cases without a fatal outcome. This is a far different picture from the situation in 1910, when Williams[61] said that a certain number would die no matter what was done.

Abortion and Miscarriage

Although it has always been accepted that about 1 in every 5 pregnancies would end in an abortion or a miscarriage, it is only recently that studies have been undertaken to determine the etiology of these events. Abortuses sent go the laboratories were treated in an evasive fashion and received scant attention; the pathologists said they were embryologic and should go to the embryologist, and the embryologists said they were pathological and should go to the pathologist. The pioneer work of Streeter[64] clarified the problem. He classified such specimens as primarily ovular, maternal or paternal in etiology. Ovular causes, having to do with defective germ plasm or faulty implantation, constitute about 70 per cent. Hertig[65] has shown that hydatid degeneration plays an important part in this group. Abortions and miscarriages due to accidents are uncommon. Because of this new knowledge it is often possible to assure a patient that loss of a pregnancy was inevitable and not due to an injudicious act on her part. Regarding habitual abortion Malpas[66] showed in 1938 that after the first event a repetition in the next pregnancy was not greater than the normal expectancy, but that the likelihood increased with succeeding gestations.

To stimulate the production of progesterone by estrogen therapy Smith[67] gave large doses of stilbestrol to a number of patients who had aborted habitually or had experienced other accidents of pregnancy. In 1948 she reported a salvage of 72 per cent in cases of early bleeding and successful prophylaxis against habitual abortion in 77 per cent. This treatment has been adopted by enough other observers to justify its use with definite hope of success.

Medical Diseases Complicated by Pregnancy

In recent years less emphasis has been placed on the possible injurious effect of pregnancy on coexisting disease, and more attention has been paid to efficient treatment of the disease itself. For this reason indications for therapeutic abortion are now infrequent.

Up to about twenty-five years ago it was believed that pregnancy definitely hastened the course of *pulmonary tuberculosis*. The general policy was to empty the uterus if the patient was first seen before the fifth month but if she was seen later, to allow the pregnancy to continue on the theory that interruption would do no good and the infant would be lost. Even in those days this seemed poor reasoning. But in 1930 the Barneses,[68] after careful study of a large number of cases, concluded that there was no difference in results with tuberculous women whether they were pregnant or not, and this opinion is generally accepted today. Therapeutic abortion, therefore, is rarely indicated in this disease.

The discovery of insulin has enabled more *diabetic women* to conceive, but it has long been known that abortion, premature labor, intrauterine death, hydramnios, congenital deformities and overgrowth of the fetus occur more often in this disease. Studies by Miller, Hurwitz and Kuder,[69] however, published in 1944, indicate that equally poor results occurred in women who had been pregnant before they became diabetic, and they reasoned that there is some fundamental factor that operates both in the diabetic and the prediabetic patient.

In 1945 White and her associates[70, 71] reported on the use of progesterone and the estrogens in diabetic patients who showed an abnormal balance involving pregnanediol and chorionic gonadotropic hormone. In such cases they obtained a fetal salvage of 96 per cent as compared with a series of similar cases not so treated, in which the salvage was only 50 per cent. On the other hand, Given, Douglas and Tolstoi,[72] without hormone treatment or strict chemical control, reported in 1950 a fetal survival of 78.6 per cent. Reis,[73] again without endocrine treatment but with careful diabetic management coupled with individualization in the time and method of delivery, obtained a fetal salvage rate of 86.4 per cent. The question of hormone treatment is still *sub judice*, but in the meantime no physician need fear that he is neglecting his pregnant patients with diabetes if he fails to use it. White reported the frequency of cesarean section in her cases as 63.2 per cent, which appears to be unnecessarily high.

Interest in the effect of pregnancy on *heart disease* in modern times began with the publication of Macdonald's[74] book on the subject in 1878. A further impetus was supplied by the contributions of Sir James MacKenzie[75] in 1921. Cardiac clinics were established at the Boston Lying-in Hospital in 1911 and at the Sloane Hospital for Women in 1913. In New England 1 woman in every 50 admitted to a maternity hospital has heart disease, which, in 95 per cent of cases, is due to rheumatic fever.

To establish a rational approach to a therapeutic plan Hamilton and his co-workers[76] undertook a study of the circulation in normal pregnancy. Their main finding was an increase in blood volume up to the ninth lunar month, which was followed by a fall in the tenth. They also found that the curve of cardiac failure coincided with that of blood volume, and they arrived at the conclusion that if a pregnant woman with heart disease could be carried to the final month of gestation the danger of cardiac failure from that point on would be greatly diminished. In cases so managed they noted that decompensation in labor was infrequent provided that the expulsive effort of the second stage was eliminated by low-forceps delivery. Cesarean section on the sole indication of heart disease was

abandoned, since it definitely increased the mortality.

The death rate in cardiac patients since the opening of Hamilton's clinic has fallen a third and is now about 2 per cent. Adherence to the rules of hygiene, diet and general medical care has increased the chance of survival four times.

In 1943 Swan and his associates[77] called attention to grave fetal defects occurring when the mother contracted *German measles* in the early months of pregnancy. These consist of cataract, deaf mutism, heart disease and microcephaly. The frequency of their appearance, depending on when the disease is contracted, is 100 per cent in the first two months, 50 per cent in the third month and 12.5 per cent after that time. Most obstetricians, therefore, consider that rubella acquired in early pregnancy is a definite indication for therapeutic abortion.

Anatomy and Physiology

When Oliver Wendell Holmes resigned the chair of anatomy at Harvard Medical School after serving thirty-five years he apologized for holding his position so long by saying that the human body was then much the same as it had been in the days of Vesalius and Fallopius. Although it may be assumed that the female reproductive tract has remained unchanged since the sixteenth century, certain discoveries made after 1900 pertain to both anatomy and physiology. One of the most important was the demonstration in 1908 by Hitschmann and Adler[78] of the progressive changes in the endometrium during the menstrual cycle. In 1917 Stockard and Papanicolaou[79] proved that these alterations could be followed in a general way by examination of the vaginal smear, and in 1928 Novak and Everett[80] found similar but less marked changes in the mucosa of the fallopian tubes.

In 1923 Painter[81] presented his work on spermatogenesis and proved that the sex of the offspring was determined biologically by the male parent, thus providing the mother with scientific ammunition when her husband is dissatisfied with the sex of the baby.

Since the beginning of the century a round dozen normal early fertilized human eggs have been found. The youngest, an implanted previllous ovum of seven and a half days (Fig. 2) was described in 1945 by Hertig and Rock.[82] The same investigators[83, 84] have also reported ova of nine and a half, eleven, twelve and twelve and a half days. Moreover, they[85] have found in the tube a two-cell ovum with a post-coital age of sixty hours, and in the uterus a morula of four days and a blastocyst of four and a half days. These are unquestionably the most important contributions to embryology of the half century.

The question of why the baby takes its first breath has been definitely settled. In 1905 Ahlfeld[86] stated his belief that the fetus *in utero* made respiratory movements. This was corroborated through animal experimentation by Barcroft[87] in 1935 and by Snyder and Rosenfeld[88] in 1937. It appears, therefore, that the infant makes respiratory movements when it is born because it has been doing so for a number of months.

All the female sex hormones have been discovered since 1900, but this is not the place to describe them. Two, however, have a particularly obstetric — albeit a somewhat academic — interest. The first is relaxin, discovered in the guinea pig by Hisaw[89] in 1929. It is derived from the corpus luteum and causes the loosening of the pelvic joints in pregnancy. Produced in excessive amounts it may create pain and disability. The second is prolactin, derived from the anterior pituitary body and described by Riddle, Bates and Dykshorn[90] in 1933. It increases the flow of crop milk in male and female pigeons and doves, it arouses the maternal instinct in virgin rats, and it induces broodiness in hens — none of which, it appears, are of enormous practical value. It is also said to augment the supply of human milk, but it is so expensive as to make its general use prohibitive.

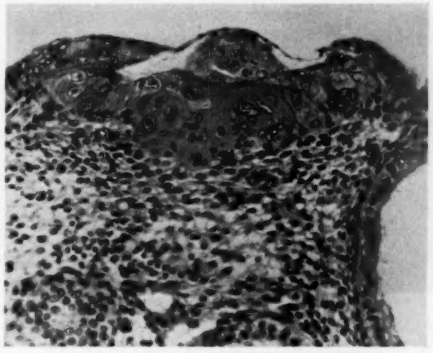

Figure 2. *The Seven-and-a-Half Day Human Ovum (Reproduced from Hertig and Rock,[83] with the Permission of the Publisher).*

Diagnosis

A useful aid to diagnosis was the classification of female pelves into four main types by Caldwell and Moloy[91] in 1933. The method of roentgenographic mensuration devised by Thoms[92] has proved simple and accurate. It enables the obstetrician to visualize the interior of the pelvis and to measure its important diameters with precision.

The most spectacular invention in obstetrics since 1900 has been the Aschheim-Zondek[93] test, which was reported in 1928. It was followed three years later by the Friedman[94] modification and by the

South African clawed-frog test, described by Shapiro and Zwarenstein[95] in 1934. All are reasonably reliable. It should be remembered, however, that the classic presumptive signs and symptoms of pregnancy still exist and that the well trained clinician is still able to make a diagnosis without resort to the laboratory.

Labor and the Puerperium

An obstetrician of 1900 would be surprised and might occasionally be alarmed by what he saw today. Having been taught that dry labor was dangerous to mother and infant, he might question the frequency of induction by rupture of the membranes, often on what appeared to be slight indications. Routine episiotomy and low forceps would seem radical, but to a colleague of the present, manual dilatation of the cervix and high forceps delivery would appear to be nothing short of assault. But when the elderly practitioner had assured himself that under proper circumstances an induced labor was not prolonged but might even be shorter than usual, that no harm came to mother or infant because of these innovations and that pelvic repairs at a later date were seldom needed, he might admit that obstetrics had improved.

Figure 3. *Boston Lying-in Hospital (Founded in 1832). A modern maternity hospital.*

Being a man of compassion he undoubtedly would approve of the steps now being taken to relieve the pains of labor. The first systematic attempt to this end was made in 1902 by Kronig,[96] who used morphine and scopolamine. When it was found that the opiate was harmful to the fetus and that scopolamine was not, a barbiturate was substituted for morphine. One of the first to use this combination was Robbins, who with his associates[97] reported his results in 1929.

In 1942 Edwards and Hingson[98] introduced continuous caudal anesthesia into obstetrics. There was nothing new about caudal anesthesia, for it had been used in a single dose for delivery by Stoekel[99] as long before as 1909. This type of pain relief possesses a definite potential danger, for the dose employed is about nine times that customarily used in spinal anesthesia. If it should inadvertently enter the spinal canal disastrous consequences, at times leading to death, may ensue.

In 1946 Read[100] advanced the hypothesis that fear is the chief cause of the pains of labor. To overcome this he uses what amounts to psychotherapy during pregnancy and delivery. He tacitly admits that his technic does not always succeed, for 5.4 per cent of his patients were delivered by cesarean section and in over a third he was forced to use accepted measures for the relief of pain. He[101] encourages the mothers to grasp their babies' hands as their bodies are being born; this distresses surgically trained obstetricians and raises the question of whether he also requires women in labor to scrub their hands just prior to the moment of birth for ten minutes by the clock. Older obstetricians who have seen many babies born before pain-relieving drugs were employed would agree that some patients suffer little in labor and that for them Read's method would serve well. Many more, however, undergo a great deal of pain, and it seems improbable that persuasion or eloquence could convince them that it does not hurt to have a baby. It is also a constant source of wonder that the vast majority of women face labor with such equanimity. If fear is the cause of pain remarkably little is said about it beforehand.

The Newborn Infant

Breast feeding has deteriorated in the last fifty years to a marked degree; in fact this is the only respect in which obstetrics has gone backward. There is evidence that infants so nourished gain more rapidly and are more resistant to disease; moreover, it encourages sociability, for Plutarch says of it that "constant feeding together is a great means to heighten the affection mutually betwixt any persons." The disinclination of many recent mothers to nurse their babies arises from many causes, but is fostered by the safety of substitute feedings — a safety not due to any pediatric magic but to the fact that most cow's milk fed to infants is pasteurized. Recently, a group of persons have appeared who are known as child psychologists. They maintain that a newborn infant should not be nursed at regular intervals but whenever it cries; otherwise, they say, it will suffer in later life a sense of frustration. How they know this they do not say. The characteristics of many American children, however, lead one to wish that there had been more instead of less family discipline, and that it had begun at the earliest possible age.

Conclusions

As one looks back over the past fifty years of obstetrics great advances, some due to laboratory discoveries and some to clinical trial and error, can be recalled. It is now five times safer to have a baby than it was in 1900, and from the baby's point of view it is twice as safe to be born. It is unnecessary to remind the reader that this has been accomplished under a system of free medicine and that it is not the result of any socialistic scheme directed by the federal Government. The cause of eclampsia, however, remains unknown; perhaps the next half century will solve the question. Physiologic studies, like those of Hamilton and his associates on the circulation, should be carried out on the other body systems to show the effects of pregnancy. New and better methods for the relief of the pains of labor will surely be found; the pharmacologist who advises a volatile liquid as safe as ether and as grateful to the patient as chloroform will be a real benefactor. But whatever the state of affairs fifty years from now, obstetricians then will undoubtedly regard those of the present day with the amiable toleration that youth reserves for age.

A typical modern maternity hospital (the Boston Lying-in Hospital) is shown in Figure 3.

References

1. National Office of Vital Statistics, Public Health Service. Personal communication.
2. Commonwealth of Massachusetts. *Fifty-ninth Report of Births, Marriages, and Deaths for the Year 1900.* Boston: Wright & Potter Co., 1901.
3. *Annual report of the vital statistics of Massachusetts; births, marriages, divorces, and deaths for the year ending December 31, 1948.* Commonwealth of Massachusetts, 1949.
4. Boston Lying-in Hospital. *Annual Report for 1949.*
5. Bacon, C. S. Mortality from puerperal infection in Chicago. *Am. Gynaec. & Obst.* **8**:429-446, 1886.
6. Reynolds, E. Cited in *The Practice of Obstetrics.* Edited by R. Peterson. 1102 pp. Philadelphia: Lea Brothers & Co., 1907. P. 941.
7. Williams, J. W. *Obstetrics: A textbook for the use of students and practitioners.* Second edition. 915 pp. New York: D. Appleton & Co., 1909. P. 874.
8. Colebrook, L., and Purdie, A. W. Treatment of 106 cases of puerperal fever by sulphanilamide (streptocide). *Lancet* **2**:1237-1242, 1937.
9. Fleming, A. On antibacterial action of cultures of penicillium, with special reference to their use in isolation of *B. influenzae. Brit. J. Exper. Path.* **10**:226-236, 1929.
10. Boston Lying-in Hospital. *Annual Reports*, 1900-1949.
11. Sänger, M. *Der Kaiserschnitt bei Uterusfibromen, nebst vergleichender Methodik der Sectio caesarea und der Porro-Operation.* 202 pp. Leipzig: W. Englemann, 1882.
12. Krönig, B. Transperitonealer, cervicaler Kaiserschnitt. In Döderlein, A. S. G., and Krönig, B. *Operative Gynäkologie.* Third edition. 955 pp. Leipzig: G. Thieme, 1912. Pp. 879-886.
13. Kerr, J. M. M. Lower uterine segment incision in conservative caesarean section. *J. Obst. & Gynaec. Brit. Emp.* **28**:475-487, 1921.
14. Latzko, W. Über den extraperitonealer Kaiserschnitt. *Zentralbl. f. Gynäk.* **33**:275-283, 1909.
15. Waters, E. G. Supravesical extraperitoneal cesarean section: presentation of new technique. *Am. J. Obst. & Gynec.* **39**:423-434, 1940.
16. Ricci, J. V., and Marr, J. P. Physick-Sellheim principle of extraperitoneal cesarean section: elucidation of technic based on 175 cases. *Am. J. Surg.* **71**:3-11, 1946.
17. Sellheim, H. Die Technik des extraperitonealen Uterusschnittes und der Entbindung durch die Uterusbauchdekenfistel. *Beitr. z. Geburtsh. u. Gynäk.* **14**:88-140, 1909.
18. Irving, F. C. Place of cesarean section. *J. Connecticut M. Soc.* **1**: 483-491, 1937.
19. *Idem.* Ten years of cesarean section at Boston Lying-in Hospital. *Am. J. Obst. & Gynec.* **50**:660-680, 1945.
20. Jansky, J. Haematologické, studie u psychotiku. (Études hematologiques, dans les maladies mentales. Rés., 131-133.) *Sborn. klin. v. Praze* **8**:85-139, 1906-1907.
21. Lewisohn, R. New and greatly improved method of blood transfusion: preliminary report. *M. Rec.* **87**:141, 1915.
22. Landsteiner, K., and Weiner, A. S. Studies on agglutinogen (Rh) in human blood reacting with anti-rhesus sera with human isoantibodies. *J. Exper. Med.* **74**:309-320, 1941.
23. Tait, L. Cited by Anspach, B. M. In *Obstetrics and Gynecology.* Edited by A. H. Curtis. 3 vol. Philadelphia: W. B. Saunders Co., 1933. Vol. 3. P. 370.
24. Schauta, F. Beiträge zur Casuistik, Prognose und Therapie der Extrauterinschwangerschaft. *Prag. med. Wchnschr.* **16**:421, 1891.
25. Dudley, A. P. Modern caesarean section, ideal method of treatment for placenta praevia. *New York M. J.* **72**:754-760, 1900.
26. Bürger, O., and Graf, R. Zur Statistik der Placenta praevia. *Monatschr. f. Geburtsh. u. Gynäk.* **25**:49-76, 1907.
27. Irving, F. C. *Outline of Abnormal Obstetrics.* 331 pp. Ann Arbor: Edwards Brothers, 1944. P. 31.
28. Findley, D. Management of placenta previa: analysis of 47,828 cases. *Am. J. Obst. & Gynec.* **36**:267-275, 1938.
29. Snow, W., and Powell, C. B. Roentgen visualization of placenta. *Am. J. Roentgenol.* **31**:37-40, 1934.
30. Ude, W. H., Weum, T. D., and Urner, J. A. Roentgenologic diagnosis of placenta previa: report of case. *Am. J. Roentgenol.* **31**: 230-233, 1934.
31. Holmes, R. W. Ablatio placentae: study based upon two hundred cases in literature. *Am. J. Obst.* **44**:752-784, 1901.
32. Couvelaire. Deux nouvelles observations d'apoplexie utéro-placentaire. *Ann. de gynéc. et d'obst.* **9**:486-495, 1912.
33. Winter. Zur Lehre von der vorzeitgen Placentarlösung bei Nephritis. *Zschr. f. Geburtsh. u. Gynäk.* **11**:398-409, 1885.
34. Fitzgibbon, G. Revised conception of antepartum accidental haemorrhage. *J. Obst. & Gynaec. Brit. Emp.* **33**:194-213, 1926.
35. Polak, J. O. Is cesarean section justifiable in ablatio placentae? *Am. J. Obst. & Gynec.* **7**:384-395, 1924.
36. Moloney, W. C., Egan, W. J., and Gorman, A. J. Acquired afibrogenemia in pregnancy. *New Eng. J. Med.* **240**:596-598, 1949.
37. Boston Lying-in Hospital. *Annual reports, 1909-1918* and *1944-1948.*
38. Bell, W. B. Pituitary body and therapeutic value of infundibular extract in shock, uterine atony, and intestinal paresis. *Brit. M. J.* **2**:1609-1613, 1909.
39. Kamm, O., Aldrich, T. B., Grote, I. W., Rowe, L. W., and Bugbee, E. P. Active principle of pituitary gland. *J. Am. Chem. Soc.* **50**: 575-601, 1928.
40. Moir, C., and Dale, H. Action of ergot preparations on puerperal uterus: clinical investigation with special reference to active constituent of ergot as yet unidentified. *Brit. M. J.* **1**:1119-1122, 1932.
41. Lever, J. C. W. Cases with puerperal convulsions, with remarks. *Guy's Hosp. Rep.* **1**:495-517, 1843.
42. Schmorl, G. Pathologisch-anatomische Untersuchungen über puerperal-Eklampsie. 106 pp. Leipzig: F. C. W. Vogel, 1893.
43. Lubarsch, O. Die puerperal Eklampsie. In *Ergebnisse der allgemeinen Pathologie und pathologischen Anatomie der Menschen und der Tiere.* Edited by Lubarsch and R. Ostertag. 8 vol. Wiesbaden; J. F. Bergmann, 1896. Vol. 1.
44. Acosta-Sison, H. Clinicopathologic study of eclampsia based on 38 autopsied cases. *Am. J. Obst. & Gynec.* **22**:35-45, 1931.
45. Davidson, J. Eclampsia and puerperal toxaemia: study of histological changes occurring in liver and kidneys. *Tr. Edinburgh Obst. Soc.* **90**:24-48, 1930-1931.
46. Fahr, T. Die pathologischen-anatomischen Veränderungen der Niere und Leber bei der Eklampsie. In *Die Eklampsie.* Edited by H. Hinselmann. 952 pp. Bonn: Friedrich Cohen, 1924. Pp. 200-292.
47. Bell, E. T. Renal lesions in toxemias of pregnancy. *Am. J. Path.* **8**:1-42, 1932.
48. Nevermann, H. Zur Bedeutung der kapillarmikroskopischen Befunde bei der Eklampsie. *Zentralbl. f. Gynäk.* **46**:617-623, 1922.
49. Hinselmann, H. Allgemeine Krankheitslehre. In *Die Eklampsie.* Edited by H. Hinselmann. 952 pp. Bonn: Friedrich Cohen, 1924. Pp. 1-87.
50. Mylius, K. Spastiche und tetanische Netzhautveränderungen bei der Eklampsie, *Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch.* **47**:379-386, 1929.
51. Smith, G. V. and Smith, O. W. Excessive gonad-stimulating hormones and subnormal amounts of oestrin in toxemias of late pregnancy. *Am. J. Physiol.* **107**:128-145, 1934.
52. *Idem.* Late pregnancy toxemia: review of experimental findings. *West. J. Surg.* **55**:288-294, 1947.
53. Tenney, B., Jr., and Parker, F., Jr. Placenta in toxemia of pregnancy. *Am. J. Obst. & Gynec.* **39**:1000-1005, 1940.
54. Reynolds, E. *Practical Midwifery: A handbook of treatment.* Third edition. 472 pp. New York: William Wood & Co., 1896. P. 269.
55. Boston Lying-in Hospital. *Annual Reports, 1945-1949.*
56. Stroganoff, V. V. My improved method of prophylactic treatment of eclampsia. *J. Obst. & Gynaec. Brit. Emp.* **30**:1-31, 1923.
57. Lazard, E. M., Irwin, J. C., and Vruwink, J. Intravenous magnesium sulphate treatment of eclampsia: collective report of 142 cases. *Am. J. Obst. & Gynec.* **12**:104-112, 1926.
58. Stander, H. J. *Textbook of Obstetrics Designed for the Use of Students and Practitioners.* 1277 pp. New York: D. Appleton-Century Co., 1945. P. 628.
59. Bryant, R. D., and Fleming, J. G. Veratrum viride in treatment of eclampsia. *J. A. M. A.* **115**:1333-1339, 1940.
60. Edgar, J. C. *The Practice of Obstetrics: Designed for use of students and practitioners of medicine.* Third edition. 1071 pp. Philadelphia: P. Blakiston's Sons & Co., 1907. P. 308.
61. Williams.[7] Pp. 508-515.
62. Irving, F. C. Treatment of pernicious vomiting of pregnancy. *Virginia M. Monthly* **67**:717-724, 1940.

63. Berkwitz, N. J., and Lufkin, N. H. Toxic neuronitis of pregnancy: clinicopathological report. *Surg., Gynec. & Obst.* **54**:743-757, 1932.
64. Streeter, G. L. Cited by Huntington, J. L. Review of pathology of 104 consecutive miscarriages in private obstetric practice. *Am. J. Obst. & Gynec.* **17**:32-41, 1929.
65. Hertig, A. T., and Edmonds, H. W. Genesis of hydatidiform mole. *Arch. Path.* **30**:260-291, 1940.
66. Malpas, P. A. Study of abortion sequences. *J. Obst. & Gynec. Brit. Emp.* **45**:932-949, 1938.
67. Smith, O. W. Diethylstilbestrol in prevention and treatment of complications of pregnancy. *Am. J. Obst. & Gynec.* **56**:821-834, 1948.
68. Barnes, H. L., and Barnes, L. R. P. Pregnancy and tuberculosis. *Am. J. Obst. & Gynec.* **19**:490-499, 1930.
69. Miller, H. C., Hurwitz, D., and Kuder, K. Fetal and neonatal mortality in pregnancies complicated by diabetes mellitus. *J. A. M. A.* **124**:271-275, 1944.
70. White, P., and Hunt, H. Pregnancy complicating diabetes: report of clinical results. *J. Clin. Endocrinol.* **3**:500-511, 1943.
71. White, P. Pregnancy complicating diabetes. *J. A. M. A.* **128**:181, 1945.
72. Given, W. P., Douglas, G., and Tolstoi, E. Pregnancy and diabetes. *Am. J. Obst. & Gynec.* **59**:729-747, 1950.
73. Reis, R. A. Personal communication.
74. Macdonald, A. *The Bearings of Chronic Disease of the Heart upon Pregnancy, Parturition, and Childbed.* 287 pp. London: J. & A. Churchill, 1878.
75. MacKenzie, J. *Heart Disease and Pregnancy.* 138 pp. London: H. Frowde, 1921.
76. Hamilton, B. E., and Thomson, K. J. *The Heart in Pregnancy and the Childbearing Age.* 402 pp. Boston. Little, Brown & Co., 1941.
77. Swan, C., Tostevin, A.L., Mayo, H., and Black, G. H. B. Congenital defects in infants following infectious diseases during pregnancy, with special reference to relationship between German measles and cataract, deaf-mutism, heart disease and microcephaly, and to period of pregnancy in which occurrence of rubella is followed by congenital abnormalities. *M. J. Australia* **2**:201-210, 1943.
78. Hitschmann, F., and Adler, L. Der Bau der Uterusschleimhaut des geschlechtsreifen Weibes mit besonderer Berücksichtigung der Menstruation. *Monatschr. f. Geburtsh. u. Gynäk.* **27**:1-82, 1908.
79. Stockard, C. R. and Papanicolaou, G. N. Existence of typical oestrous cycle in guinea pig — with study of its histological and physiological changes. *Am. J. Anat.* **22**:225-283, 1917.
80. Novak, E., and Everett, H. S. Cyclical and other variations in tubal epithelium. *Am. J. Obst. & Gynec.* **16**:499-530, 1928.
81. Painter, T. S. Studies in mammalian spermatogenesis. II. Spermatogenesis of man. *J. Exper. Zoöl.* **37**:291-321, 1923.
82. Hertig, A. T., and Rock, J. Two human ova of pre-villous stage, having developmental age of about seven and nine days respectively. *Contrib. Embryol.* (No. 200) **31**:65-84, 1945.
83. *Idem.* Series of potentially abortive ova recovered from fertile women prior to first missed menstrual period. *Am. J. Obst. & Gynec.* **58**: 968-993, 1949.
84. *Idem.* On development of early human ovum, with special reference to trophoblast of previllous stage: description of 7 normal and 5 pathologic human ova. *Am. J. Obst. & Gynec.* **47**:149-184, 1944.
85. Hertig, A. T. Personal communication.
86. Ahlfeld, F. Die intrauterine Tätigkeit der Thorax und Zwerchfellmuskulatur, intrauterine Atmung. *Monatschr. f. Geburtsh. u. Gynäk.* **21**:143-163, 1905.
87. Barcroft, J. Respiratory and vascular changes in mammal before and after birth. *Lancet* **2**:647-652, 1935.
88. Snyder, F. F., and Rosenfeld, M. Direct observation of intrauterine respiratory movements of fetus and role of carbon dioxide in their regulation. *Am. J. Physiol.* **119**:153-166, 1937.
89. Hisaw, F. L. Experimental relaxation of pelvic ligaments of guinea pig. *Physiol. Zoöl.* **2**:59-79, 1929.
90. Riddle, O., Bates, R. W., and Dykshorn, S. W. Preparation, identification and assay of prolactin — hormone of anterior pituitary. *Am. J. Physiol.* **105**:191-216, 1933.
91. Caldwell, W. E., and Moloy, H. C. Anatomical variations in female pelvis and their effect on labor, with suggested classification. *Am. J. Obst. & Gynec.* **26**:479-505, 1933.
92. Thoms, H. Uses and limitations of roentgen pelvimetry. *Am. J. Obst. & Gynec.* **34**:150-152, 1937.
93. Aschheim, S., and Zondek, B. Schwangerschaftsdiagnose aus dem Harn (durch Hormonnachweis). *Klin. Wchnschr.* **7**:8, 1928.
94. Friedman, M. H., and Lapham, M. E. Simple, rapid procedure for laboratory diagnosis of early pregnancy. *Am. J. Obst. & Gynec.* **21**:405-410, 1931.
95. Shapiro, H. A., and Zwarenstein, H. Rapid test for pregnancy on Xenopus laevis. *Tr. Roy. Soc. South Africa* **22**:75, 1933-1934.
96. Krönig, B. Scopolamine-morphine narcosis in labour. *Brit. M. J.* **2**:805-808, 1908.
97. Robbins, A. R., McCallum, J. T. C., and Zarfas, L. G. Use of sodium iso-amylethyl barbiturate (sodium amytal) in obstetrics. *Am. J. Obst. & Gynec.* **18**:406-415, 1929.
98. Edwards, W. B., and Hingson, R. A. Continuous caudal anesthesia in obstetrics. *Am. J. Surg.* **57**:459-464, 1942.
99. Stoekel, W. Über sakrale Anästhesie. *Zentralbl. f. Gynäk.* **33**:1-15, 1909.
100. Read, G. D. Correlation of physical and emotional phenomena of natural labour. *J. Obst. & Gynaec. Brit. Emp.* **53**:55-61, 1946.
101. *Idem.* Discomforts of childbirth. *Brit. M. J.* **1**:651-654, 1949.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 37031

PRESENTATION OF CASE

A fifty-five-year-old woman entered the hospital because of vomiting and right-upper-quadrant abdominal pain.

The patient stated that for about five years before admission she had attacks of right-upper-quadrant pain, at times associated with nausea and vomiting. Her doctor presumed this to have been gall-bladder disease, but no x-ray films had been taken. Her health was fairly good until five days prior to entry, when the right-upper-quadrant pain became more severe than usual and pain was also present in the epigastrium. This persisted until the day before entry, and on two occasions was so severe that the patient required morphine for relief. Once or twice daily, during this five-day period, she vomited greenish material that was presumed to be bile. There was no history of fever, chest pain, cough or other significant symptoms. She stated that the night before admission she felt chilly but had no true shaking chill. During this history taking she seemed very drowsy and answered questions poorly.

She had nocturia (two to three times) for some years that was increasing in frequency lately, but she had no marked polyuria or dysuria, hematuria or pyuria. She was thought to have asymptomatic hypertension, and had been enormously obese all her life. Her father had had diabetes, and had died with renal disease; her mother had died of heart disease. Two siblings were living. A son had diabetes.

Physical examination showed an obese woman, with an enormous pendulous abdomen surmounted by an easily reducible umbilical hernia. (The hernia was described by one examiner as incarcerated.) The skin was dry, scaly and discolored. There was a faint odor of acetone to the breath, and the mucous membranes were dry. The patient breathed through the mouth. The lungs were clear. The heart was not enlarged. There was a soft apical systolic murmur and a diastolic gallop best heard at the third intercostal space 2 cm. to the left of the sternum. The abdomen was huge, and difficult to palpate. No definite visceral masses or tenderness could be elicited. One examiner could hear no peristalsis. Neurologic examination was negative.

The temperature was 98.2°F., and the pulse 105. The blood pressure was 110 systolic, 68 diastolic.

The urine had a specific gravity of 1.022 and a pH of 6, and gave a red reaction for sugar and a ++ reaction for acetone; the sediment contained 1 to 2 granular casts per high-power field. Examination of the blood disclosed a hemoglobin of 15.1 gm. and a white-cell count of 23,100, with 90 per cent neutrophils. The blood sugar was 566 mg., and the nonprotein nitrogen 40 mg. per 100 cc., and the carbon dioxide 7 milliequiv., the potassium 2.9 milliequiv., the sodium 125 milliequiv., and the chloride 85 milliequiv. per liter. A stool was guaiac negative. The electrocardiograms were thought to be compatible with a low serum potassium. A plain x-ray film of the abdomen was reported as unsatisfactory. There was a single gas-filled loop of distended bowel—apparently colon — in the left lower abdomen, which suggested distended sigmoid. A tube was present in the upper abdomen, lying far to the left and anteriorly.

The patient vomited 600 cc. of "engine oil" material that gave a positive guaiac test. She was immediately given 50 units of regular insulin intravenously and 40 units subcutaneously, and 500 cc. of physiologic saline solution intravenously. She was placed on "coma therapy." In the following ten hours she received 2500 cc. of physiologic saline solution and 3500 cc. of 5 per cent dextrose in water intravenously, which contained 4 ampoules of potassium solution and 2 ampoules of sodium lactate. She also received 340 units of insulin. After eight hours the blood sugar was 364 mg. per 100 cc., and the carbon dioxide 21.9 milliequiv. per liter. The urinary sugars were green in the presence of blood sugar levels in excess of 400 mg. per 100 cc. The urine output was small, with a volume of 400 cc. in ten hours. The nonprotein nitrogen was 40 mg. per 100 cc. at the start of therapy. The potassium was 3.9 milliequiv. per liter, fell to 2.0 milliequiv., and then rose to 2.6 milliequiv. per liter. The hematocrit was 46 per cent eleven hours after entry.

After twelve hours of therapy, the patient was little improved, except for better hydration. At 3:00 a.m. on the morning following admission, the vital signs were stable and at satisfactory levels, with adequate urine output, when suddenly the blood pressure fell to 80 systolic, 40 diastolic, and the respirations became deeper at the rate of 26 per minute. The heart rate continued regularly at 105, with a gallop rhythm. Only small amounts

of urine could be obtained in the final hours of life. The patient had nystagmus. By the time a blood transfusion was started, the nail beds had a cyanotic hue, and the respirations were becoming slower. She died seventeen and a half hours after admission to the Emergency Ward.

During the last hours of life the blood sugar was 290 mg., and the phosphorus 1.3 mg. per 100 cc.; the carbon dioxide was 21.7 milliequiv., the sodium 132.9 milliequiv., and the potassium 3.3 milliequiv. per liter.

Differential Diagnosis

Dr. James H. Townsend*: This case is an appropriate one to be discussed in Diabetes Week, since it draws attention to a situation that we are trying so hard to prevent through education of people to detect early diabetes in themselves and of physicians to recognize dangerous symptoms before they have advanced to irreversible stages.

There are few cases on which the Medical Service works harder than those of diabetic acidosis, for we believe that we should approach a 100 per cent record of success unless there is disease that would be fatal in itself. Any fatal case evokes general discussion of why and how it might have been handled differently if it could have been done again.

Interest in this case centers on trying to explain why this patient, who entered the hospital with all the usual symptoms and signs of severe diabetic acidosis (drowsiness, hyperpnea, dehydration, glycosuria, hyperglycemia, ketonuria and markedly reduced carbon dioxide and pH), and who seemed to be doing pretty well by the usual criteria of disappearance of sugar and ketones in the urine, falling blood sugar, rising carbon dioxide, a well maintained blood pressure, and fair urine output, should have taken a turn for the worse and died after fourteen hours of therapy.

There may be many reasons that separately or together account for such a patient's failure to respond to reasonable therapy. There may have been a condition present that might of itself be fatal, or in which the chemical disturbance tipped the scales against recovery, which might have taken place were it not present. There may have been unsuspected defects in the cardiovascular system, including the kidneys — particularly in the older age groups and in patients with diabetes of long standing. The therapy may have been inadequate in insulin or in other important aspects. The condition of acidosis, with its cumulative losses of water and electrolytes, and interference with important cellular functions, may have been allowed to go on so long before recognition that irreversible changes in vital cells had taken place. One never wishes to accept this possibility as long as the patient is still breathing, but there must be a "point of no return" somewhere, so that even when the best known therapy is given, failure will result.

Let us look at the available evidence in this case from these points of view. Was disease present other than the diabetes that caused death or contributed materially to it? I strongly suspect that there was, but I see no way to prove it from the available evidence, or to decide what it was. That may come out in Dr. Castleman's denouement. Several possibilities suggest themselves. The fact that the acute illness was initiated by severe abdominal pain requiring morphine suggests strongly that the pain was due to a pathologic condition in the abdomen, rather than the pain of acidosis, which is usually a late symptom appearing after the patient has already shown other symptoms of acidosis, such as marked thirst, weakness and hyperpnea. The patient was too sick to give a clear history of the order of development of her symptoms. She had had previous attacks of apparently similar pain, thought to be due to gallstones, which it may well have been. This abdomen must have been very unsatisfactory to examine. No localized tenderness or spasm was found — these are usually the most important points in the notoriously difficult differential between the abdominal pain of acidosis and pain due to an acute surgical condition. When seen by the surgical service on admission, the patient was apparently conscious enough to respond to painful stimuli. Even so, I think it is impossible to rule out acute cholecystitis with rupture and peritonitis. The white-cell count of 23,000 helps not in the least — it is so common in acidosis alone. Of the conditions that cause recurrent attacks of upper abdominal pain, it is the most common.

Another possibility, which has been not too infrequently found in fatal diabetic coma, is acute pancreatitis, yet there is no evidence on which I can base this diagnosis. Nothing is said about back pain. One would expect tenderness. There is a note by one observer that no peristalsis was heard, suggesting peritonitis from some cause. Among the many laboratory tests I do not find an amylase determination.

The patient had an umbilical hernia. One examiner described it as "incarcerated"; another examiner described it as "easily reducible." The surgeons apparently made little of it, yet if it had really been incarcerated for five days there could have been gangrenous gut. The pain is not described as colicky, but again I do not have a very clear description. There was no general gaseous distention. Perhaps at this point we might see the x-ray films, which were taken with this possibility in mind. I doubt that there was intestinal obstruction of five days' duration, but again I cannot rule it out.

Dr. James J. McCort: The x-ray films are not available.

Dr. Townsend: In diabetic acidosis, once the stomach has been evacuated by tube, fluid given

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slowly will usually be tolerated. In this case the patient did not vomit any more, and she took 450 cc. in the fourteen-hour period, according to the policy of oral feeding as early as possible, — especially foods containing potassium and phosphorus, — but just before death 700 cc. was aspirated, 250 cc. more than she took in. There may have been even more there. Dilatation of the stomach is a common event in acidosis. Again, this suggests abdominal disease interfering with normal peristalsis, but this is not too unusual in severe acidosis in which no intra-abdominal disease is found. On the whole, I am inclined to the opinion that the patient did have an acute intra-abdominal condition, most likely connected with the gall bladder.

What about the cardiovascular system? There were no neurologic signs pointing to a cerebral lesion. There was no direct evidence of heart disease per se, except possibly the gallop rhythm. I have not seen enough hypopotassemia to know whether or not this is a common feature of it. Coronary-artery disease is notoriously common in diabetic women, but the electrocardiograms taken five days after the acute illness began are not reported to show any evidence of a myocardial infarction.

As to the kidneys — two examinations of the urine sediment showed casts, commonly found in diabetic acidosis, but no pus. This would seem to rule out a urinary-tract infection, which is so common in diabetic women. The nonprotein nitrogen of only 40 mg. per 100 cc. after five days of a dehydrating illness argues strongly against any important degree of organic kidney defect. The urine output was only fair — 430 cc. in fourteen hours. She probably did have interference with renal function, of the anoxic type seen in severe diabetic acidosis, but I doubt that much organic change will be demonstrated, although autopsy may well show glycogen infiltration of the tubules.

Does the disappearance of sugar from the urine while the blood sugar level was over 500 mg. per 100 cc. indicate kidney disturbances? I think not, but I think it does denote long-standing diabetes. The acetone also disappeared from the urine very quickly, and was never present in large amounts. We occasionally see patients in severe diabetic acidosis who have little or no ketone bodies in the urine, when there have been large amounts of ketones in the blood, as indicated by a simple test that uses acetone powder with serum several times diluted. It usually occurs with severe oliguria, and I have supposed it meant inability of the kidneys to excrete ketones. The test was apparently not done in this case.

Pneumonia is frequently found post mortem in such cases. However, the lungs were clear clinically, she had no fever, and she had received prophylactic penicillin. The possibility of pulmonary embolism should be mentioned, but it seems unlikely.

The therapy was certainly adequate, according to most commonly accepted standards, but there is no other field in which there is such heated debate over details. The patient received 340 units of insulin in fourteen hours. The blood sugar dropped from 566 to 290 mg. per 100 cc. Roughly 45 gm. of sugar, in addition to the 175 gm. she received intravenously, was either stored or utilized, for she did not excrete any appreciable amount. There is no evidence that more insulin would have helped her.

As to water and electrolytes, she received intravenously in fourteen hours 6380 cc. of water; 646 milliequiv. of sodium, or 101 milliequiv. per liter; 520 milliequiv. of chloride, or 81 milliequiv. per liter; 120 milliequiv. of potassium, or 19 milliequiv. per liter; 48 milliequiv. of phosphorus, or 8 milliequiv. per liter; and 198 milliequiv. of lactate, or 31 milliequiv. per liter. The ratio of mineral cations to anions was 1.3. The ratio of sodium to chloride was 1.24. There was an excess of 1380 cc. of water over a solution isotonic in electrolytes. This seems to me a very reasonable regimen.

Butler,[1] in his recent review in the *New England Journal of Medicine*, gives his method of estimating the needs in the first twenty-four hours of therapy, but it is not clear to me how to translate the needs of a child weighing 30 kg. into those of this fifty-five-year-old, very obese woman. If, however, one followed in general his suggestions about an initial hydrating fluid — assuming 1500 cc. in this case, followed by his potassium Solution C with magnesium, and assuming the use of 5 ampoules in 5000 cc. of fluid — making a total intravenous volume of 6500 cc., it would include 425 milliequiv. of sodium and 25 milliequiv. of magnesium (in other words, somewhat less sodium and chloride, no more potassium, a little more phosphorus, and 25 milliequiv. of magnesium). These differences do not impress me as great.

The striking thing in the chemical determinations is the low potassium from the very beginning — 2.9 milliequiv. per liter before death. The electrocardiograms were thought to be consistent with hypopotassemia. Potassium in the serum is usually high at the start of therapy, reflecting rapid release of potassium from cells, falling to lower levels four to six hours later under the influence of insulin. To me this low value at the start indicates very severe cellular depletion incident to the five-day acute illness. The patient may have been losing potassium even before this. Could this have been corrected by more intensive potassium therapy? Perhaps, but it would have involved the use of solutions more concentrated than those commonly considered safe.

Guest,[2] in his review in the *American Journal of Medicine* in November, 1949, advises the use of 1 gm. of potassium chloride per hour for 4 doses

when low potassium is found. That would be only about 50 milliequiv. This woman had more than twice that amount. In a few cases some physicians have used an isotonic or stronger solution of potassium salts, and there is a recorded case in which as much as 467 milliequiv. of potassium chloride was given in eighteen hours without ill effect, but such procedures cannot be said to be free from danger. Perhaps this patient was one on whom such therapy might have been used, but that is pure speculation. She did have hypopotassemia, and may have died of its effects.

We have no exact knowledge about the use of magnesium. Magnesium determinations are very hard to obtain, particularly in an emergency, and I think there is no way of diagnosing magnesium deficiency clinically, even though we may suspect it.

This patient certainly had hypophosphatemia. The final value was 1.3 milliequiv. per liter. I do not know what the initial value was. However, we do not know of any definite ill effects of this. It is another indication of the severe state of cellular electrolyte depletion.

She received 175 gm. of glucose, but it was not given until the urine was essentially sugar free and the blood sugar was definitely falling. It certainly did not cause excessive diuresis. It may have contributed to the passage of potassium out of the serum, but probably no more than the insulin itself. I am not told whether she received oxygen. Its use seems logical. There seemed to be no clear indication for transfusions of blood or albumin in the early hours, since the blood pressure was quite well maintained — never going below 100 systolic until shortly before death, and rising as high as 140 systolic, 70 diastolic.

It should be emphasized that the carbon dioxide level bears little relation to the prognosis in such cases. The duration of the acidotic symptoms is far more important, with its accompanying cellular depletion of electrolytes, and disruption of such things as ATP (adenosine triphosphate), and other complex organic phosphates that have to do with phosphorylation and other enzymatic reactions in cells. Some investigators have reported the direct injection of such substances as ATP or thiamine pyrophosphate, with apparently good results. This is in the realm of research, but it may be practicable, and it is certainly simple to use vitamin B preparations in the fluid to be given intravenously, since thiamine and riboflavin and perhaps others are concerned as coenzymes, and may be seriously depleted in cases like this.

Another thing to remember about the carbon dioxide level is that at least two elements contribute to its reduction: first, the accumulation of organic and ketone bodies in the serum, and second, the reduction in circulating cations because of the losses of sodium and potassium, which continue progressively as the acidosis continues. In this case the amount of circulating ketones may have been relatively small, and it appears that the loss of alkali, both cellular and extracellular, was the important thing. The initial sodium of 125 milliequiv. per liter was about 15 milliequiv. deficient; this in itself would account for the reduction of carbon dioxide to 12 milliequiv.

In summary, I would list my diagnoses as follows: diabetes mellitus with severe acidosis of long duration, severe electrolyte depletion with hypopotassemia and acute intra-abdominal disease — acute cholecystitis being the most likely.

Dr. Reed Harwood: I should like to congratulate Dr. Townsend on a masterly discussion of this difficult problem. I saw this patient after five or six hours of treatment. At that time she was conscious and was able to answer questions well. I examined the abdomen and could find no tenderness or spasm. She had the most enormous, pendulous abdomen that I have ever seen, and I imagine there was very little muscle in the pendulous portion that could have been expected to show spasm. It is my opinion that she died of potassium deficiency, in spite of what was found in the abdomen at postmortem examination. I base this on the fact that in the past we have had a number of other patients who were not expected to die, but who petered out at the end of ten to eighteen hours, although progress up to that time had seemed satisfactory. During the last few hours this patient's blood pressure dropped to 80 systolic, 40 diastolic, a sign that in the past we have learned to consider of ominous prognostic significance. The respirations went up. Whether there was anything that could be done is doubtful, of course. My opinion is that she would have died eventually of the abdominal condition, since it is unlikely that the correct diagnosis would have been made. She died in the critical period of diabetic coma, I believe from potassium deficiency.

I wonder if we could have prolonged her life had we given her more blood? I think the transfusions should have been started at the time that the blood pressure, particularly the diastolic pressure, began to fall. Another question comes to mind. In this case we had determined what the serum potassium was from the very beginning. The flame photometer was working, and the electrocardiograph was working — the evidence was all there that she was not getting an overdose of potassium parenterally. Perhaps Dr. McArthur would have an opinion about whether this was a case in which we could really have pushed potassium.

Dr. Janet W. McArthur: I think you are quite right. I think this was really a death from low potassium, although it is very difficult to say. I think we could have given her more potassium.

Dr. Townsend: I should think it was the kind of situation in which up to 400 or 500 mg. of potassium could be given fairly rapidly, controlled by the

electrocardiogram and frequent potassium determinations.

DR. HARWOOD: In the last two years most of our patients have had electrodes for the electrocardiogram on at all times. It is quite interesting to watch the T waves come down as therapy progresses.

DR. JOHN T. STANBURY: Was a terminal potassium done on this patient?

DR. HARWOOD: It was 3.6 milliequiv. per liter.

DR. STANBURY: Dr. Richard Goldsmith and I, here in the back row, have been muttering about the severity of the diabetes, and wondering whether the abdominal catastrophe broadly hinted at just now by Dr. Harwood was not the leading cause in this patient's death, aided and abetted by a mild diabetes, accentuated by the severe infection, and with the acidosis augmented by starvation in an obese woman. After all, the initially normal potassium could be interpreted as the end result of severe depletion, as Dr. Townsend suggests, or as a figure not yet shifted from normal, because the acidosis was early and mild. There is little here to suggest long-term severe depletion, and recovery in terms of carbon dioxide content was prompt. We venture to suggest that diabetes was an important, if secondary, contributing cause of death in this patient.

DR. HARWOOD: I think it is most unlikely that the low carbon dioxide content would be caused by starvation alone. I think Dr. Townsend expressed it very well when he stated that there were several things that contributed to death, and that the abdominal condition did not make the situation any better.

CLINICAL DIAGNOSES

Diabetes mellitus, with acidosis.
Low potassium syndrome.

DR. TOWNSEND'S DIAGNOSES

Diabetes mellitus, with acidosis.
Hypopotassemia.
Acute disease in the abdomen, ?acute cholecystitis.

ANATOMICAL DIAGNOSES

Acute appendicitis, with perforation and abscess formation.
Diabetes mellitus (with acidosis).
Obesity, marked.
Umbilical hernia.
Pulmonary edema.

PATHOLOGICAL DISCUSSION

DR. BENJAMIN CASTLEMAN: The abdominal condition was an acute appendicitis. The right colon and the cecum were rotated in such a way that they were located in the epigastrium. The appendix was 16 cm. long, and its last 2 to 3 cm. was quite purple and gangrenous, and had set up a series of small abscesses in the abdomen. If the patient had been operated upon, the surgeons would have had a hard time finding and draining them. The abdomen was really huge. The hernia came down to about the knees of the patient. There was severe edema of the lungs, so that I wonder whether further transfusions would have been of value. The liver was quite large, and showed a great deal of intracellular fat. Of course she was such a large person — she weighed about 350 pounds, I would guess — that I do not know how much of the liver enlargement was due to her size. The pancreas showed very severe hyalinization of the islands of Langerhans, so that it is certainly true that she must have had the disease for quite a while. The kidneys were large, but showed no evidence of infection or intercapillary glomerulosclerosis.

A PHYSICIAN: Did you say the hernia was incarcerated?

DR. CASTLEMAN: It was not incarcerated.

REFERENCES

1. Butler, A. M. Diabetic coma. *New Eng. J. Med.* **243**:648-659, 1950.
2. Guest, G. M. Diabetic coma: metabolic derangements and principles for corrective therapy. *Am. J. Med.* **7**:630-646, 1949.

CASE 37032

PRESENTATION OF CASE

First admission. A thirty-three-year-old man was admitted complaining of nausea and vomiting of three days' duration and of weakness.

One month prior to admission the patient noted gradually increasing weakness, easy fatigability and general malaise. Three weeks before admission a submaxillary abscess developed from a periodontal infection. This was drained externally, and two teeth were extracted in the Out Patient Department. Physical examination at that time was negative. The blood pressure was 160 systolic, 100 diastolic. Urinalysis was negative for sugar but showed a trace of albumin and 2 to 3 red blood cells, and 2 to 3 white blood cells per high-power field in the sediment. Three days previous to admission he developed anorexia, epigastric pain, nausea and later vomiting. In the month before admission there was some polydipsia and polyuria, and weight loss of 20 pounds.

Physical examination revealed a well developed, thin, debilitated man. There was a small draining sinus at the angle of the left mandible. Examinations of the heart, lungs, abdomen and central nervous system were negative.

The temperature was 99°F., the pulse 80, and the respirations 20. The blood pressure was 118 systolic, 78 diastolic.

Examination of the blood showed a hemoglobin of 15 gm. and a white-cell count of 9650, with 68 per cent neutrophils. Urinalysis showed an orange

sugar reaction and a + test for acetone; the sediment contained no cells. The fasting blood sugar was 310 mg., the nonprotein nitrogen 15 mg., and the cholesterol 318 mg. per 100 cc., and the carbon dioxide 16.5 milliequiv. per liter.

The diabetes was readily controlled, and the patient was discharged after thirteen days on a program of 12 units of protamine zinc insulin every morning and a diet containing 200 gm. of carbohydrate, 100 gm. of fat and 75 gm. of protein. The dental infection and draining sinus were healed by the time of discharge.

Second admission (two years later). In the interim he had carried on well on the above regime until eight days before this admission, when he developed diarrhea with 7 to 8 loose, watery stools during the day. The diabetes was out of control, but he was easily regulated on insulin by urine tests. He was discharged after eight days on a regimen of diet and insulin therapy similar to the previous one, with the addition of 6 units of regular insulin before breakfast as judged by the morning urine test.

Third admission (eight years later). In the interval he had been followed closely in the Diabetic Clinic. He had only a few temporary mild upsets in control, usually in relation to infections, until one year before this admission. The diet had been increased to 300 gm. of carbohydrate, 150 gm. of fat and 110 gm. of protein, with 20 to 24 units of protamine zinc insulin, to satisfy his requirements as a manual laborer. Two years before admission he had become impotent. One year before admission he was admitted to the Emergency Ward for post-dental-extraction cellulitis. This responded readily to antibiotics, and control was rapidly regained. Following this he noted slowly progressive weakness of the legs, associated with numbness of the toes and fingers. Examination showed impaired vibration sense below the level of the thighs, definite motor weakness of the dorsiflexors of the feet, absent ankle and knee reflexes and diminished pinprick sensation in the left calf. He was treated with vitamin B complex and crude liver extract, with some subjective improvement. Seven months before admission, pyoderma of the scalp developed. In spite of routine therapy, including antibiotics, this persisted, with the development of small abscesses, which had to be drained. The last of these had been drained two weeks before admission, but instead of improving the infection had extended, necessitating admission to the hospital. In addition he complained of diarrhea of one week's duration, with 2 to 3 watery stools per day.

Physical examination revealed a large granulating wound in the right temporo-occipital region with an area of fluctuation posterior to it. Neurologic examination was as described above. No other abnormalities were found.

The temperature was 99.3°F., the pulse 90, and the respirations 20. The blood pressure was 140 systolic, 80 diastolic.

Examination of the blood showed a white-cell count of 11,200. Urinalysis disclosed a specific gravity of 1.025, a negative reaction for sugar and a + test for albumin. The fasting blood sugar was 101 mg. per 100 cc. Barium-enema examination was negative, and x-ray films of the skull normal. Proctoscopic examination showed a spastic, hyperemic colon, with no masses or ulcers. The stools were guaiac negative. The scalp infection cleared rapidly with wet dressings, débridement and antibiotics. The patient was discharged on the seventh day on a regimen of diet and insulin as before. At the time of discharge he was having 3 to 4 soft stools a day.

Final admission (four months later). In the interval he continued to have intermittent bouts of diarrhea with 0 to 1 stool during the day and 3 to 4 at night. The stools were described as loose and semiformed, without blood, mucus or tarry material. These bouts were not associated with pain, chills, fever, nausea or vomiting. The diabetes had remained well controlled. He had become very depressed because of his inability to work and the necessity of relying on charitable agencies and his family for support. He became nervous and easily annoyed. He had a few attacks of paralysis that lasted several days; these were diagnosed by his physician as attacks of hysteria. Two days before admission he began to have generalized cramping abdominal pain, which was partially relieved by vomiting. These pains were associated with shaking, sweating and weakness. He vomited many times during the two days and was unable to take any food. He continued to take insulin daily. The urinary sugar reaction was orange during the night before admission. There was a bowel movement, reported to be normal, the night prior to admission.

On physical examination the skin was warm and perspiring. Respirations were Cheyne-Stokes in type. There were coarse crackles and rales at the right apex and at both bases. The heart appeared to be of normal size. The sounds were of fair quality. The rhythm was regular, but there was a protodiastolic gallop. There were no murmurs or thrills. There was no abdominal distention, and no abdominal tenderness or spasm. Peristalsis was present, but appeared diminished.

The temperature was 100.6°F., the pulse 120, and the respirations 30. The blood pressure was 100 systolic, 80 diastolic.

Examination of the blood revealed a hemoglobin of 16 gm. and a white-cell count of 14,000. A catheterized specimen of urine had a specific gravity of 1.020 and gave a green reaction for sugar and a negative test for acetone. The sediment contained innumerable red cells, an occasional white cell and no casts. The blood sugar was 600 mg., and the

nonprotein nitrogen 63 mg. per 100 cc.; the sodium was 130.0 milliequiv., the potassium 5.5 milliequiv., the chloride 90 milliequiv., and the carbon dioxide 19.0 milliequiv. per liter.

Following admission the patient continued to vomit periodically a dark-brown, watery material. He was given 25 cc. of 50 per cent glucose intravenously, followed by 200 cc. of 10 per cent dextrose in water and 1000 cc. of physiologic saline solution. Eight hundred cubic centimeters of fluid material was aspirated from the stomach, including food ingested two days previously. Following this the blood pressure was 70 systolic, and he received 250 cc. of plasma. In the next three hours he was given another unit of plasma and 500 cc. of physiologic saline solution. He was started on insulin by test, and had received 135 units of regular insulin by the end of the second hospital day. During this day urine output was 830 cc. and vomitus 1860 cc. The fluid intake was 5680 cc. This consisted of 680 cc. taken by mouth and the remainder given intravenously, in the form of 500 cc. of plasma, 1500 cc. of 5 per cent dextrose in water, 1500 cc. of 5 per cent dextrose in physiologic saline solution with potassium ion (Solution B), and 1500 cc. of dextrose in physiologic saline solution alone.

During the second hospital day the nonprotein nitrogen was 92 mg., and the sugar 720 mg. per 100 cc.; the carbon dioxide was 22.4 milliequiv., the sodium 133.5 milliequiv., the chloride 95 milliequiv., and the potassium 5.4 milliequiv. per liter. The pH was 7.22. In the evening the patient was noted to be acutely dyspneic, with distended neck veins. Both lungs were full of moist rales. The heart rate was rapid, with a gallop. The blood pressure was 96 systolic, 88 diastolic. The temperature was 101.4°F., the pulse 106, and the respirations 36. The patient was given 0.8 mg. of Cedilanid intravenously and 50 mg. of Demerol subcutaneously. An electrocardiogram showed supraventricular tachycardia (rate of 115). The P waves were not seen. The ST segments were depressed in Leads V_4 and V_5. The QT interval was normal.

On the morning of the third hospital day the patient's condition was unchanged. An x-ray film of the chest was taken, and on his return to the ward he was restless, dyspneic and orthopneic. In spite of emergency resuscitative measures he died ten minutes later. Oxygen had been supplied via nasal tube during the journey to the Radiology Department.

Differential Diagnosis

Dr. Janet W. McArthur*: May we begin by seeing the x-ray films?

Dr. James J. McCort: The barium-enema examination performed on the third admission discloses no gross defects. The wide folds suggest mucosal edema, but there is no evidence of mucosal ulceration. A faint homogeneous density throughout both lung fields is seen on the chest film taken on the final admission. This diffuse homogeneous density is compatible with pulmonary edema. In addition, there is a mottled density in the right upper lobe, as well as in the superior segment of the right lower lobe, which has the appearance of a pneumonia. Fluid is present in the right base, blunting the right costophrenic sinus. The transverse diameter of the heart is slightly increased, without a characteristic configuration. On the plain film of the abdomen made on the last admission a drainage tube is seen in the stomach. There is no small-bowel or large-bowel dilatation. The kidneys are within normal limits as to size and shape, and there is no evidence of calcification in the course of either urinary tract.

Dr. McArthur: Is the appearance of the lungs suggestive of nephritic edema, or is there just ordinary diffuse pulmonary edema?

Dr. McCort: I think it is a diffuse pulmonary edema, with a suggestion of superimposed pneumonitis in the right upper lobe.

Dr. McArthur: To give a thumbnail sketch, this was a patient whose diabetes was first recognized when he was thirty-two years of age, who suffered a long series of infections and neurologic complications following the onset of the diabetes, and who came to an abrupt and untimely end at the age of forty-one. The terminal picture was that of shock and what I take to be congestive heart failure. Since this is Diabetes Week I suppose I am obliged to look for a moral in this case. If there is a moral here, it is that the diabetic person who takes poor care of himself runs the risk of being plagued by neuropathy. The evidence that relates most diabetic complications to poor control of the disease is not altogether convincing; however, in his monograph Rundles[1] makes out a good case for such a connection with respect to diabetic neuropathy.

Two years after diabetes was first detected, the patient began to have diarrhea; and this dogged him ever afterward. Gradually the diarrhea acquired the noctural timing characteristic of "diabetic diarrhea," whatever that is. The lower gastrointestinal tract was thoroughly studied during the third hospital admission by means of proctoscopy, barium enema and examination of the stools. Since these disclosed nothing of note, I think I am entitled to interpret the diarrhea as a manifestation of visceral neuropathy. Six years after the onset of diabetes the patient developed impotence, another indication of diabetic neuropathy. Unlike the sexual disturbances in diabetic women, which appear to have a hormonal basis, the sexual disorders of diabetic men are primarily neurologic in origin. Eight years after the diabetes was first recognized, there were manifestations of peripheral neuropathy,

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with weakness of the legs, numbness, loss of deep tendon reflexes and impaired vibration sense. This complication apparently followed hard upon an acute infection — a cellulitis of the jaw — which required treatment in the Emergency Ward. The timing of this fresh complication illustrates another point made by Rundles. A new manifestation of neuropathy is likely to appear immediately after a bout of acidosis or an acute infectious upset.

In the course of these eight troubled years the patient became anxious and depressed because of his incapacity and his need to rely on charitable agencies. Then, five days before death, he was suddenly seized with cramping abdominal pain and vomiting, which went on for two days before he was admitted to the hospital. It was noted on admission that he had no localized abdominal pain, spasm or distention, and therefore I am immediately going to rule out all such entities as acute pancreatitis, mesenteric vascular occlusion and acute intestinal obstruction. Although he was admitted in critical condition, the diabetes was very well controlled, presumably because he had continued to take insulin at home despite the vomiting. He was slightly acidotic, the serum carbon dioxide content being 19 milliequiv. per liter, but he was not in diabetic coma, and I do not think I can validly attribute the abdominal symptoms to acidosis. To correct the electrolyte disturbances that resulted from vomiting, he was given heroic therapy with physiologic saline solution. The role played by the fluid therapy in this case is difficult to assess, but it is hard to see how it could have been beneficial.

After the initial phase in the hospital, the vomiting ceased, and more ominous indications appeared. The patient became dyspneic, rales could be heard throughout both lungs, and a protodiastolic gallop rhythm became audible. Finally, he began to exhibit signs of shock, the systolic blood pressure falling to 70. At the time of death there were indications, not only of left ventricular failure, but of right ventricular failure as well, since the neck veins became distended. The problem, then, really centers around the etiology of the terminal shock and congestive failure. I obtain no help from the history in the form of antecedent heart disease. No murmurs were ever heard, there were no symptoms that could be interpreted as angina pectoris, and the blood pressure was found to be elevated on only one occasion.

There are not many conditions that will bring the heart to precipitous failure under these circumstances. The one that comes first to mind is acute nephritis. This man had red and white cells in the urine at the time he was first seen, he was noted to have albuminuria of a slight degree off and on afterward, and finally the urine was loaded with innumerable red cells. He appears to have had a latent nephritis, which underwent a severe terminal exacerbation. However, I doubt that this lesion sufficed to bring him to heart failure and death.

I should like to mention another condition — one that might readily produce such a clinical picture in a diabetic patient — namely, acute myocardial infarction. From the autopsy studies of Root and Graybiel,[2] we know that all diabetic patients, who have had their disease for more than five years show some degree of coronary arteriosclerosis. Moreover, roughly 10 per cent of all diabetic deaths are due to myocardial infarction. While the clinical appearance presented by the patient is entirely compatible with such a diagnosis, the electrocardiogram is disappointing. Dr. Edwin O. Wheeler, who has kindly reviewed the description of the tracing, assures me that the changes are nonspecific in character. The depression of the ST segments in Leads V_4 and V_5 could be due solely to the fact that the patient had been given Cedilanid. One would expect to see distinctive electrocardiographic abnormalities following a myocardial infarct so extensive as to produce congestive failure and death. However, it is well known that in exceptional cases the electrocardiographic patterns diagnostic of myocardial infarction are tardy in making their appearance.

In conclusion, I am going to say that this man had diabetes mellitus with visceral and peripheral neuropathy, a latent glomerulonephritis that became acute at the time of death, and coronary atherosclerosis — with a question of acute myocardial infarction. He may have had some degree of intercapillary glomerulosclerosis, but I have no real clinical warrant for making that diagnosis.

DR. MCCORT: I should have mentioned, for whatever it is worth, that there is a slight sclerosis of the pelvic arteries in this patient.

DR. MCARTHUR: That makes me feel a little better.

DR. JAMES H. TOWNSEND: I am inclined to believe that since albumin in the urine appeared over the years, he probably did have, along with his generalized vascular disease, a considerable degree of intercapillary glomerulosclerosis. I am also quite impressed with the elevated nonprotein nitrogen. I believe he will be found to have had considerable kidney damage.

DR. REED HARWOOD: I would agree with you — and expect to find some intercapillary glomerulosclerosis present. Unfortunately, we never examined the ocular fundi, but I would be willing to bet that he also had a considerable degree of retinitis.

DR. TOWNSEND: The relation between neuropathy and vascular disease does not seem very clear. In general, the neuropathy in most patients appears to be a reversible process that they can get over in time, but the same people who have it are likely to have vascular disease also. Whether there is a common underlying basis or whether they are both due to neglect of the diabetes I do not think we know, but they are likely to be encountered in the same patient.

CLINICAL DIAGNOSES

Diabetes mellitus.
Pneumonia.
Congestive failure, with pulmonary edema.

DR. MCARTHUR'S DIAGNOSES

Diabetes mellitus, with visceral and peripheral neuropathy.
Coronary sclerosis, with ?acute myocardial infarction.
Acute exacerbation of glomerulonephritis.

ANATOMICAL DIAGNOSES

Acute myocardial infarction.
(*Diabetes mellitus, with neuropathy.*)
Bronchopneumonia.
Pulmonary edema.

PATHOLOGICAL DISCUSSION

DR. BENJAMIN CASTLEMAN: The autopsy here proved that Dr. McArthur was correct. This patient did die of heart failure, the heart disease being a severe coronary sclerosis with a recent thrombus in the right coronary artery, producing acute myocardial infarction of the entire posterior wall of the heart, extending over into the septum. There was, in addition, evidence that he probably was having infarctions of the heart over a considerable period before admission, because there were healed and healing infarcts present that might have been two to three weeks old, in contrast to the recent one, which looked four or five days old. I think there is no doubt that he was having infarcts for a fairly long period before the recent one. The lung showed a very severe degree of edema and, as Dr. McCort said, showed also an area of early bronchopneumonia. There were some old fibrotic changes in the apexes, with emphysema and chronic pneumonitis. The other organs were not remarkable.

DR. MCARTHUR: What did the kidneys show?

DR. CASTLEMAN: The kidneys were normal.

REFERENCES

1. Rundles, R. W. Diabetic neuropathy: general review with report of 125 cases. *Medicine* **24**:111-160, 1945.
2. Root, H. F., and Graybiel, A. Angina pectoris and diabetes mellitus. *J. A. M. A.* **96**:925-928, 1931.

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THE PLAN

NOTHING conduces more to panic than the contemplation of disaster, and the lack of a plan by which to meet it. Such has been the situation in this country in recent years, with a population and a leadership apparently scarcely struggling against the strange inertia that seemed to grip them. Conditions have changed, however; a real emergency at last confronts the civilized world, and the possibility of a premeditated assault even on this country has suddenly become not so remote.

If the attack on the industrial centers should come, the salvation of the citizen who survives it in a relatively uninjured condition will be to know that he has work to do and what that work is, in order that as many as possible of the injured may also survive. Obviously, something resembling order can be created in the midst of chaos only through a well developed plan that gives to each available worker the job that he must do if circumstances in any way permit.

Such a plan for Massachusetts has been presented by Currier and Bradford in this issue of the *Journal*. Without going into specific details it outlines the essential principles of a basic defense program that needed only the breath of authority to give it life.

That authority has now been issued in the form of Publication AG-11-1 of the Federal Civil Defense Administration, on Health Service and Special Weapons Defense, and the organization of personnel in each community, each district, each hospital can be underway, the storage of essential supplies and provisions can begin, hospital liaisons can be arranged and perfected and a mass movement of defense can be set in motion.

Uncertain of the part it should play in shaping world destinies, this country has perhaps again demonstrated its unreadiness for world leadership. It must not, however, show for a moment longer unreadiness for its own protection. Plans are being made and the majority of the people are ready and eager to follow them.

RESPONSIBILITY FOR HOSPITAL STANDARDIZATION

SIR HENEAGE OGILVIE* maintains that surgery attracts the "doers" rather than the "thinkers" in medicine. The surgeon's interests, he believes, are humanitarian rather than scientific, and the guiding circumstance of his life is personal responsibility. He wants to be in command, to make decisions and to direct the lines of action that such decisions may involve.

Although many surgeons cannot be fitted to this mold and others might object to being placed in it, there is much to support Sir Heneage's views. It may be that these qualities account for the corporate action of the American College of Surgeons when it first assumed the responsibility for hospital standards in 1915. Now, after thirty-five years of distinguished service and accomplishment, the College is faced with a grave decision concerning

*Ogilvie, H. Surgeon's life. *Lancet* 2:1-6, 1948.

this responsibility. The story is brief, and it is one with which the entire medical profession should be familiar.

In 1918, when the American College of Surgeons rendered its first report on hospital standardization, only 12 per cent of the 692 institutions surveyed met the comparatively simple standards initially stipulated. Today, 96 per cent of all hospitals of more than 100-bed capacity meet the very rigid requirements set for the adequate care of the sick. In support of this humanitarian venture the College has donated over $2,000,000 from the dues of its fellows.

Occasionally, over the years, it has been questioned whether such a comparatively small segment of the profession should assume and bear the complete responsibility for hospital standards. More recently, the extraordinary financial burden entailed by this task, nearly 4000 hospitals having been surveyed in 1949, has been a concern to the regents of the College. Many of them have believed that these funds could be used to greater advantage in an expansion of the graduate training program. On the other hand, the fellows and officers of the College and many of the beneficiaries of the hospital standardization program are loath to see so grave a responsibility relinquished to less experienced hands.

The matter came to a head this year, when the American Hospital Association, which has long maintained an interest in the problem, suggested to the regents of the American College of Surgeons that it would be willing to take over the responsibility and financial cost of hospital standardization. This offer specified that the control of professional and hospital standards would be delegated to a commission of 25 members, of whom 3 would be physicians and 3 surgeons. The rest of the commission was to be made up of hospital trustees and administrators.

This offer of the American Hospital Association was carefully studied and rejected at a meeting of the Board of Regents on August 4, 1950. Unfortunately, however, on the following day, the Board of Trustees of the American Hospital Association adopted a resolution to establish a hospital standardization program. This action led to considerable confusion, since it was interpreted by many as an indication that the American College of Surgeons had relinquished its responsibility for hospital standards. This was not and is not the case.

The regents of the College have announced that it will maintain and expand its program of hospital standardization even at the cost of deficit financing until such time as a co-ordinated group representative of the entire medical profession can be established for the maintenance of the greatly expanded program that will eventually be required. To this end a number of conferences with representatives of the American Medical Association, the American Hospital Association and the American College of Physicians have been held. A final solution has not been reached, but much progress has been made.

Although the American Hospital Association has much to offer in this matter, it is obvious that the final responsibility should be a professional and not an administrative one and should be shared by the entire profession. Undoubtedly, a final and happy solution to the problem will be forthcoming. Until then, however, it is good to know that the humanitarian instincts and keen sense of personal responsibility that Sir Heneage regards as characteristic of surgeons will keep them at the task of hospital standardization just as they were led to it in the first place.

"THE GIFT AND THE GIVER"

THE thirty-second annual report of the Commonwealth Fund includes, in addition to the usual description of the activities of the Fund, a tribute to the late Mrs. Edward S. Harkness, till recently honorary president, ending with the words, "And great good came from the gift and the giver." The phrase epitomizes the way in which, through the years, the Fund has achieved the purpose of its founder: "To do something for the welfare of mankind." Its contribution to science in particular and to mankind in general is illustrated by its activities in support of medical education and research, experimental health services, public health and hospital practice.

In the field of medical education appropriations totaling $693,370 went to Boston University School

of Medicine for support of a home-care program, to University of Louisville School of Medicine for teaching of mental health in pediatrics and public health and to Memorial Hospital of New York, Western Reserve University School of Medicine and Washington University School of Medicine (Division of Psychosomatic Medicine) for educational programs; the advanced fellowships in medicine and allied fields received continued support. Public-health and hospital activities included grants of more than $550,000, with emphasis on consultation service in a medical center in Hunterdon County, New Jersey, extension of the home-care plan at Montefiore Hospital, continuation of field and educational services from a former public-health program, hospital construction and operating expenses for the Council of Rochester Regional Hospitals and continuation of a former rural-hospital program. For medical research at various institutions, $380,000 was allocated. More than $200,000 was contributed to the Commonwealth Fund fellowships, including expenses of administration and operation in England. Miscellaneous activities, consisting of grants to the American Psychiatric Association, the Group for the Advancement of Psychiatry, the Institute of International Education and the World Federation for Mental Health, accounted for nearly $115,000.

This record of private support of projects in fields vital to the progress of medicine and public health is heartening, in spite of the fact that the amounts appropriated are small in comparison with those available from the federal Government. The Commonwealth Fund continues to rank high among the organizations that fulfill the purposes of public-spirited philanthropy. As in previous years, the gift has encouraged scientific endeavor of great worth, and the wisdom and generosity of the giver have set an example to those who work for the advancement of science in a free society.

CAUDAL APPENDAGE

THE *British Medical Journal*, among its medical memoranda published on October 14, 1950, reports the relatively rare curiosity of an eleven-month-old child with an authentic caudal appendage. The tail in question was something more than rudimentary, if considerably less than fully functional. Capable of controlled motion, it could be gently wagged, if indeed not lashed, although it possessed few if any of those prehensile characteristics that would permit the owner to swing from branch to branch. A bit of evidence in favor of the descent of man, it was totally inadequate to break his fall when that descent became too swift. When pulled out to its full length it measured over 3 inches: when not in use "it would contract, curl on itself, resembling the tail of a pig, and then relax and flick from side to side." There being no statement that might suggest any psychosomatic relation, it is inferred that the tail's owner neither purred when it flicked nor barked when it wagged.

Despite the insistence of the late William Jennings Bryan and others to the contrary, evidence accumulates that man either descended or rose, depending on the point of view, from less gifted animals, and the case in point lends substance to the theory.

Some confusion, but not much, is added to the discussion by consideration of the taillessness of the cats of the Isle of Man in view of the fact, rendered immortal by the famous college reunion song, that all the other cats have tails in England, Ireland, Scotland, Wales. The apparent injustice of it is tempered by a sensible submission to the divinity that shapes men's ends.

Anything is charlatanism which is morally dishonest, though it may be practised under cover of a diploma; and therefore that man is a charlatan, to all intents and purposes, who, like the notorious Restell, becomes the executioner of babes in utero.

Boston M. & S. J., *January 15, 1851*

MASSACHUSETTS MEDICAL SOCIETY

DEATH

STUART — Frederick W. Stuart, M.D., of Boston, died on December 25. He was in his ninety-first year.

Dr. Stuart received his degree from Harvard Medical School in 1884. He was formerly a member of the staff of the Boston Dispensary, New England Medical Center.

A daughter, a son, six grandchildren and two sisters survive.

OBITUARY

PAUL GUSTAFSON, M.D.

1899–1950

Dr. Paul Gustafson was born in Arlington, of Swedish parents, the third in a family of nine. He later resided in Cambridge, where he attended the local schools and Harvard College, graduating in 1912. An academic record of honors in chemistry and allied subjects was supplemented by an unusual record in athletics. Always an outstanding lacrosse player, he led Harvard to a mythical national championship in that sport in 1912, and kept up his interest as a player and coach of lacrosse for many years thereafter.

Before graduation from Harvard Medical School in 1916, he had enrolled in the Harvard Surgical Unit for service with the British troops in France in World War I. He served overseas two and a half years on the staff of General Hospital No. 22, B.E.F., under Colonel Hugh Cabot. During this service he showed the alertness, efficiency and devotion to duty that made him outstanding in his profession in later years.

On return from France in 1919, he served as surgical intern at the Boston City Hospital and obstetric intern at the Boston Lying-in Hospital, followed by an internship in gynecology at Sloane Hospital for Women in New York during 1921.

At the time of his death on November 25, 1950, he was senior obstetrician at the Boston Lying-in Hospital and chief obstetrician at Mount Auburn Hospital, Cambridge. He was a certified member of the American Board of Obstetrics and Gynecology, the American Medical Association, the Massachusetts Medical Society, the Cambridge Medical Improvement Society, the American College of Surgeons, the New England Obstetrical and Gynecological Society, the New England Pediatric Society, and the Obstetrical Society of Boston, of which he had been president in 1948. He was also an instructor in obstetrics at Harvard Medical School.

At various times he had been consulting obstetrician at the Winchester Hospital, Emerson Hospital, Concord, Milton Hospital, Brockton Hospital, Nashua, New Hampshire, Hospital, and Woonsocket, Rhode Island, Hospital. He was formerly assistant obstetrician at the Massachusetts General Hospital.

He belonged to the Harvard Club of Boston, the Country Club and the Exiles, a doctors' social group. He was interested in many social and charitable projects and gave liberally toward their support. Dr. Gustafson was unmarried, and had made his home for many years with a sister, Miss Emma Gustafson.

It is with great sorrow that his medical associates, his many patients, and the community to which he contributed so much record the passing of Dr. Gustafson, a warm friend and counselor who gave unselfishly of his time and energy wherever and whenever he could be of service.

F. P.

NOTES FROM THE MEDICAL EXAMINER*

THE LAW OF MISSING PERSONS

The necessity of establishing the "corpus delicti" in a criminal prosecution is well known to everyone. However, few are cognizant of the problems identification of the dead creates in civil procedures.

In order to transfer interests in jointly held stocks and bonds or to pay the beneficiary of a life-insurance policy, a death certificate is usually required by the very terms of the contracts. A medical examiner cannot make such a certification if the body is missing or cannot be properly and accurately identified. Yet a presumption of death is strongly implied when persons disappear in conflagrations, train and airplane accidents, maritime disasters and drownings even though the body is not recovered after a thorough search. Such disappearances are becoming more and more prevalent and create litigious problems regarding the disposal of the property of the missing person.

A person interested in this estate must choose one of the following legal procedures. If he decides that the circumstances reasonably warrant believing the person dead, he can proceed to probate the estate, using the date of disappearance as the date of death. Probate courts do not require a death certificate, but any other interested person can challenge the fact of death during the course of probation and quash the proceedings. Thus, it is practical to approach first the insurance companies and obtain their consent to probation. If this request is refused, one would have to bring a bill in equity or an action in contract and establish death by a "reasonable certainty." This requires the affidavits or the testimony of the "res gestae witnesses" to the circumstances surrounding the disappearances, as well as proof of an adequate but fruitless search for the "corpus." If the court is convinced that death may be safely presumed, the insurance company or the corporation would be compelled to transfer the interest to the proper custodian for purposes of settling the estate. This is the standard of death by "reasonable certainty" as established by the Massachusetts courts in Sargent's case.[1]

Should the disappearance of the person be under such circumstances that death cannot be established with "reasonable certainty" the person is presumed living for a statutory period of fourteen years. During this time his property is preserved

*Presented by the Department of Legal Medicine, Harvard Medical School.

by a receiver appointed by the probate court.[2] Allowances and adjustments to provide for the widow and minors of the missing person will be made, and at the end of this statutory period the presumption of death becomes legally established. The act of the court in probating the estate under the laws of intestacy is final in one year. Even though the "missing person" reappears after twelve months he cannot challenge the court for denial of his property.

Identification of unknown bodies by a proper medicolegal investigation can often avoid this cumbersome statutory period before death can be presumed. The diligent use of photographs, dental charts, fingerprints and x-ray films, together with the careful notation of scars, moles, tattoos, anomalies, malformations and prior operations, as well as preservation of all clothing of unknown bodies, is strongly urged. The mystery of a missing person so solved will often eliminate an unpleasant, procrastinating legal procedure.

References

1. Sargent's Case, 307 Mass. 246.
2. Chap. 200, Annotated Laws of Mass., Settlement of Estates of Absentees.

Woodrow W. Weiss, M.D., LL.B.
Research Fellow, Department of Legal Medicine
Harvard Medical School

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

PROGRAM FOR ALCOHOLIC PATIENTS

In an act passed on August 17, 1950, the General Court authorized the Department to develop a program for alcoholism. The legislation was supported by the Commission on Alcoholism, which had been studying the problem since January, 1948, and by various interested groups of individuals and organizations in the Commonwealth.

Objectives

The objectives assigned to the Department by the Act are threefold: the establishment of such clinic and hospital facilities as are necessary for the proper diagnosis, treatment and rehabilitation of persons addicted to the excessive use of alcoholic beverages; the study of problems related to alcoholism; and the promotion of a preventive and educational program concerning the problems of alcoholism.

In carrying out these objectives, the Department is directed to consult and work in conjunction with recognized scientific and service organizations and other agencies actively interested in and dealing with problems of alcoholism.

Division of Alcoholism

By authority of the legislation embodied in Chapter 800 of the Acts of 1950, the Department has established the Division of Alcoholism, which began its activities on November 16, 1950, under the direction of John C. Ayres, M.D. A general advisory committee composed of representative citizens of the Commonwealth with an active interest in alcoholism has been appointed. Under the guidance of this committee a program is being outlined. Policies, which are being set up, are based on recommendations made by recognized authorities in the field of alcoholism and on experiences of similar programs already in effect throughout the country. Because much is yet to be learned about how best to operate such programs, policies will remain flexible and subject to change with the dictates of experience.

Basis of Program

The program of the Department is being planned with full recognition of the fact that alcoholism is an illness that can be treated successfully even though with great difficulty. Highest priority is being assigned to treatment and rehabilitation. Research can be carried along with treatment and service to the patient. As for education and prevention, there is no finer medium than the recovered alcoholic addict.

FLUORIDATION OF PUBLIC WATER SUPPLIES

The Public Health Council has recently approved the following policy on the fluoridation of public water supplies:

> The Massachusetts Department of Public Health will approve, upon application from local Boards of Water Commissioners with the endorsement of City Councils or Boards of Selectmen, the fluoridation of public water supplies as a partial dental caries control measure. The plans for adequate hydraulic, mechanical and laboratory controls must be submitted to the Department for approval and provisions made for adequate pre-fluoridation and post-fluoridation dental evaluations. The Department will provide technical assistance in establishing recommended controls and evaluation procedures to local Boards of Water Commissioners and Health Departments through the Divisions of Sanitary Engineering and Dental Health.

This policy has been adopted because the Department believes that sufficient evidence has been accumulated to indicate that the procedure produces valuable results. At present, all potable Massachusetts waters may be considered deficient in fluorine. Three auxiliary water supplies show concentrations of fluorine approaching the optimum but are of little significance, being available only occasionally to a few families.

The Department believes, therefore, that all Massachusetts communities may properly investigate the feasibility of fluoridating their potable waters.

CORRESPONDENCE

DR. LOUIS MENDELSOHN

To the Editor: No person who ever knew Dr. Louis Mendelsohn would be satisfied to see him ushered out of the land of the living with only a routine obituary as a sendoff. He was so much the man and the doctor, and had so large a coterie of sincere friends, and so many leaned upon his strength, or depended upon his advice and leadership, that his unusually productive career warrants special consideration.

Dr. Mendelsohn was born in Sweden in 1889, coming as a boy to Boston, where his father opened a small grocery in the West End. He grew up in Cambridge and entered Harvard in the class of 1901, joining the Pierian Sodality as violinist. He graduated from Harvard Medical School in 1904, his violin having helped to finance his education.

He served his internship in Carney Hospital; on graduating he married Miss Marjorie Piers, of an old Philadelphia family, and took a house on Washington Street, Dorchester, where he opened his office. He later moved his office to Boston and his home to Brookline, where he organized a string quartet, which practiced chamber music regularly every two weeks. He served for years in the medical Out Patient Department of the Massachusetts General Hospital.

Dr. Mendelsohn was a man of high integrity, sincere, sensitive and strict in his ethical standards; the competitive pressure and commercial attitudes that he sometimes encountered in his professional contacts preyed upon him so that he considered seriously giving up his well established and lucrative practice and moving to more congenial territory.

In 1922 he consequently went with his wife to California, determined to select a location in that state. A few weeks of investigation led them to Saratoga in the foothills of the Coast Range, overlooking the Santa Clara Valley. A few years before, the striking possibilities of this region as a place of residence had inspired several San Francisco businessmen to build their houses on narrow ledges above the canyons. They were soon followed by retired or retiring professors, musicians, churchmen, Army and Navy officers and artists, from various sections of the country, who settled on lower and more convenient levels.

These people needed and could appreciate a friendly, scholarly, and competent physician, and Dr. Mendelsohn fitted the need to perfection. He purchased the practice of the retiring physician, and had constructed an attractive medical building, which soon found itself in a community center, for the library, the town tennis court (of both of which he was a trustee) and the women's club building were promptly erected close by. In 1929 he was elected president of the Saratoga Chamber of Commerce.

His reputation grew with his practice, patients coming to him from over a wide area, and soon he was appointed to the staff of the Stanford University Hospitals in San Francisco. He built himself a large bungalow in a two-acre apricot orchard, and erected a slathouse in which to experiment with various subtropical fruits not yet naturalized. He reconstituted his quartet, which for several years participated freely and effectively in constructive communal efforts.

One of the beautiful estates in Saratoga is Montalvo, a classical structure with a long terraced garden as beautiful as any on the Riviera, built by a connoisseur of art, Senator Phelan. When the Senator died he left this estate, with its notable paintings and statuary, to the San Francisco Art Museum on the condition that Dr. Mendelsohn be made curator of the real estate, and that it be employed for promoting community interest in the fine arts. This led to the organization of the Montalvo Society, which has ever since provided the people of the area with programs of high cultural content.

After his death in November the board of supervisors of Santa Clara County voted to rename a portion of the old Saratoga — Los Gatos Road, Mendelsohn Lane, in his honor.

ALBERT EHRENFRIED, M.D.

Brookline, Massachusetts

"UNFAIR TO ORGANIZED LABOR"

To the Editor: In the editorial entitled "The Mountain Labors" in the November 9 issue of the *Journal*, I think the author is quite unfair about the attitude of organized labor toward the American Medical Association. Labor is interested in developing a national health program as proposed by President Truman in Senate Bill 1679, Eighty-First Congress. It asks for a fair debate upon the merits of the program. It believes that the Association, by its direct and tie-in advertisements and by the use of other tactics, is not making an honest effort to discuss the program, but is trying to confuse the public regarding just what the Truman National Health Program calls for. Organized labor believes that the public is entitled to know about this attitude of the Association toward a fair presentation of the facts.

To accomplish this end in addition to other methods organized labor uses the labor press. If the author of the editorial mentioned above does not approve of the statements in the labor press, may I ask if he thinks the statements put out in the medical and lay press by organized medicine are more edifying?

A few examples among the many of how the Association over the years has spread misinformation about proposed legislation in favor of a national health program to be financed in part by compulsory health insurance are as follows:

> In 1947, when the National Physicians Committee was opposing a bill in the Eightieth Congress similar to S. 1679, the American Medical Association supported the statements of the National Physicians Committee, although published reports of the Association's own committee showed that numerous statements of the National Physicians Committee were false.
>
> In opposing the present S. 1679, the Association, in its so-called educational campaign by the use of such terms as "socialized medicine" and "welfare state" and by spreading misinformation regarding the details of what the bill calls for, builds up a picture of a program quite contrary to what the proposed legislation contains, and then proceeds to attack this imaginary program.
>
> The Association has repeatedly misrepresented what is happening in England and in claiming that the English plan is a failure has implied at the same time that S. 1679 calls for a similar program. This, of course, the Association knows is not the fact.
>
> Finally, the educational campaign of the Association sank to an all time low when it supported the trash put out by a group of organized medicine in Maryland entitled "Waiting Room Willie."

Can one blame labor for being a bit rough but still honest when the Association sets such an example?

Labor knows that many of its members are not receiving in a satisfactory manner the excellent medical care available in this country. Labor favors a plan that it honestly believes will improve the delivery of medical care. It asks for a free discussion of the subject. The Association, instead of accepting the challenge and discussing the problem on its merits, has undertaken a propaganda campaign in which much misinformation has been broadcasted.

There are many conscientious and well informed physicians who are ashamed of the approach of the Association to the discussion of this controversial question and believe that the attitude of organized labor is more dignified than that of the Association.

CHANNING FROTHINGHAM, M.D.

Boston

BOOK REVIEWS

Essays on a Science of Mythology: *The myth of the divine child and the mysteries of Eleusis*. By C. G. Jung and C. Kerényi. Translated by R. F. C. Hull. 8°, cloth, 289 pp., with 5 illustrations. New York: Bollingen Foundation, Incorporated, 1950. $4.00. (Bollingen Series 22.)

These essays first appeared in the form of two smaller books: the *Divine Child* (1940) and the *Divine Maiden* (1941), published in occupied Amsterdam. The present translation includes a new Prolegomena and Epilegomena by Kerényi. The essays are literary excursions into mysticism and have nothing to do with science, as Kerényi himself points out (p. 5). Jung, on the other hand, argues that his is the method of natural science, since he describes his observations and classifies them (p. 218); yet even he regards science as a

"living and lived myth, satisfying to persons . . . cut off from their psychic origins by neurotic dissociation" (p. 136). The entire book has a poetic quality and a strong flavor of medieval scholasticism. Kerényi's exposition is oblique and neglects to make explicit many of his most elementary notions, and Jung communicates efficiently only with himself.

Dealing with the Divine Child, Kerényi discusses a number of common ideas, or "mythologems," which appear in certain Finnish, Hindu and Greek accounts of the childhood of the gods. For example, several of these deities were not born but "emerged from primal waters"; others were "quite at home in the primal water." Again, writing of the Divine Maiden, this author is impressed by the resemblance between the Greek Persephone and a maiden goddess in the mythology of a nonliterate culture on the Polynesian island of Ceram: both these figures descend in to the earth periodically and are somewhat seasonal about their reappearance, like grain. Jung discusses the symbolic meaning of the Child and associates the content of some of his patients' fantasies with a number of mythical events. To account for the associations, he offers a résumé of his theory of archetypes in the "racial unconscious."

The central thesis of this book is built around the appearance of similar events and symbols in the myths of different cultures. Despite the extremely esoteric nature of many of these parallels, both authors are dogmatic in their exclusion of all possible explanations for them other than their own basic hypothesis. This hypothesis asserts that common myth-forming ideas are present in the "unconscious psyche" of all persons and are *therefore* independent of learning in individual experience. This logic will not convince many thoughtful readers. Psychologists and ethnologists, in particular, will remain unimpressed.

The Envelope: A study of the impact of the world upon the child. By James S. Plant, M.D., Sc.D. 8°, cloth, 299 pp. New York: Commonwealth Fund, 1950. $3.00.

This book, written by a psychiatrist, shows the successful influence of interdisciplinary interests and training. One finds here a mature integration of the various human behavior sciences as they impinge on the problems of personality growth and development.

The core of this treatment revolves around the concept "envelope," which refers to a mental screening device employed by the person to select only facts and phenomena that have meaning to the personality. The remaining 23 chapters of this book are devoted to relating this concept to the development of personality.

One finds throughout the book a healthy eclecticism regarding psychiatric theory, including some questioning of the author's own assertions. By way of illustration one might point to the critique of concepts such as rejection and insecurity. Here Dr. Plant attempts to delineate the areas of behavior that such concepts describe and goes on to suggest operational definitions.

Many questions are raised regarding the relation between sociocultural factors and personality development. At these points Dr. Plant interjects some refreshing insights. For example, he suggests that the number of persons in a household is of little concern in attempts to understand personality development. More important than the number of persons in the household is the character of relationships among these persons.

One cannot read this book without being impressed by the literary and psychologic profundities. "Adjustment is not so much a matter of attainment as of acceptance of the fact that one is always looking for it."

The limitations of this treatment are centered around the absence of a more objective quantitative treatment in the handling of data. Although Dr. Plant does present the crystallization of his clinical experiences, one cannot help feeling at points that the function of the "envelope" has also left its imprint regarding what the author chose as being significant. One must admit, however, that Dr. Plant has tried to develop a systematic theory regarding personality development, and certainly one that should attract the empirically oriented scientists of human behavior interested in the testing of theoretical formulations.

This book should make excellent reading for the physician in general practice and is certainly essential for those concerned with the problem of a theory of personality.

NOTICES

ANNOUNCEMENTS

Dr. John J. Kneisel announces the opening of his office for the practice of general surgery at 1 Greenridge Avenue, White Plains, New York.

Dr. Daniel Miller announces the removal of his office for the practice of otolaryngology to 403 Commonwealth Avenue, Boston.

JOSEPH H. PRATT DIAGNOSTIC HOSPITAL

Conference Program

Tuesday, January 23 — Polycythemia: Primary and secondary. Dr. Ulfar Jonsson.

Wednesday, January 24 — Triethylene-melamine (TEM) in the Therapy of Hematological Disorders. Dr. Jay Silverberg.

Friday, January 26 — Physical Medicine in the Treatment of Nervous Disorders. Dr. Arthur L. Watkins.

Tuesday, January 30 — X-ray Conference. Dr. Alice Ettinger and Staff.

Wednesday, January 31 — Endocrine Clinic. Dr. Edwin B. Astwood.

Conferences are held in the Pratt Lecture Hall.

All exercises are open to the medical profession.

CUTTER LECTURE

The Cutter Lecture on Preventive Medicine will be held in Amphitheater E of the Harvard Medical School on Friday, February 9, at 5 p.m. Dr. Hugh M. Sinclair, professor of nutrition at the University of Oxford, England, will speak on "Nutrition Surveys of Population Groups."

All physicians and medical and public-health students are invited.

FELLOWSHIPS IN TUBERCULOSIS

The closing date for filing applications for fellowships in tuberculosis has been extended to February 1, 1951. These awards, administered by the National Research Council for the National Tuberculosis Association, are designed to promote the training and development of investigators in this field. They are open to citizens of the United States who are graduates of American schools and who, as a rule, are not more than thirty years of age. Preference will be given to applicants who have the degree of doctor of medicine or doctor of philosophy. Only in exceptional circumstances will a fellowship be awarded to an applicant without either of these degrees.

Inquiries should be addressed to the Secretary, Medical Fellowship Board, National Research Council, 2101 Constitution Avenue, N. W., Washington 25, D. C.

SOCIETY MEETINGS AND CONFERENCES

January 19 and 20. National Research Council, Committee on Human Reproduction. Page 1066, issue of December 28.

January 22. Boston Lying-in Hospital Obstetric Round-Table Conference. Page 629, issue of October 19.

January 23. Norfolk District Medical Society. Page 629, issue of October 19.

January 23. Hampden District Medical Society. Page 74, issue of January 11.

January 23-31. Joseph H. Pratt Diagnostic Hospital. Notice above.

January 25. Boston City Hospital House Officers' Association. Page 74, issue of January 11.

January 29. Suffolk District Medical Society. Page 37, issue of January 4.

January 31. American Academy of Orthopaedic Surgery (see Kappa Delta Award for Research in Orthopedic Surgery). Page 736, issue of May 4.

February 8. Leukemia. Dr. Joseph Ross. Pentucket Association of Physicians. 8:30 p.m. Haverhill.

February 9. Beta Iota Chapter of Nu Sigma Nu. Page 1018, issue of December 21.

(*Notices concluded on page xiii*)

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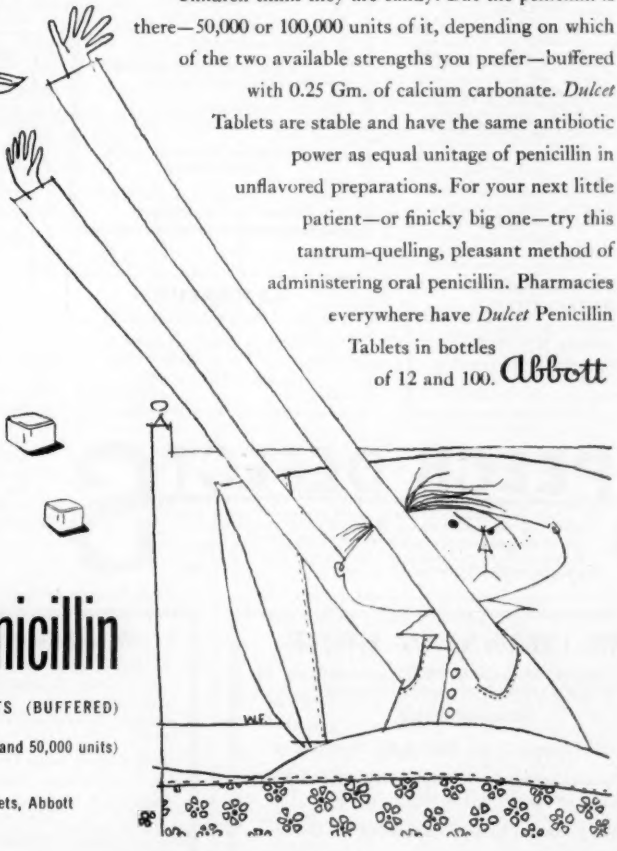

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NOTICES (*Concluded from page 116*)

FEBRUARY 9. Cutter Lecture. Page 116.
FEBRUARY 11. National Conference on Medical Services. Page 38, issue of January 4.
FEBRUARY 12–14. American College of Allergists. Page 74, issue of January 11.
FEBRUARY 13. New England Society of Anesthesiologists. Page 512, issue of September 28.
FEBRUARY 21. Bay State Society for the Crippled and Handicapped. Page 1066, issue of December 28.
FEBRUARY 27. Norfolk District Medical Society. Page 629, issue of October 19.
MARCH 5–8. New Orleans Graduate Medical Assembly. Page 1018, issue of December 21.
MARCH 7. Hampshire District Medical Society. Page 170, issue of July 27.
MARCH 11–16. College of Medical Evangelists. Page 74, issue of January 11.
MARCH 22. Hyman Morrison Lecture. Page 74, issue of January 11.
MARCH 26. Boston Lying-in Hospital Obstetric Round-Table Conference. Page 629, issue of October 19.
MARCH 27. Norfolk District Medical Society. Page 629, issue of October 19.
APRIL 9–13. American College of Physicians. Page 296, issue of June 15.
APRIL 10. New England Society of Anesthesiologists. Page 512, issue of September 28.
APRIL 24. Norfolk District Medical Society. Page 629, issue of October 19.
MAY 8. New England Society of Anesthesiologists. Page 512, issue of September 28.
MAY 9. Norfolk District Medical Society. Page 629, issue of October 19.
MAY 16. New England Obstetrical and Gynecological Society. Hillcrest Country Club, Leicester, Massachusetts.
May 22–24. Massachusetts Medical Society. Annual Meeting. Hotel Statler, Boston.
MAY 24–26. American Goiter Association. Columbus, Ohio.
SEPTEMBER 19. American Medical Writers' Association. Page 1066, issue of December 28.
SEPTEMBER 19–21. Mississippi Valley Medical Society. Page 1066, issue of December 28.
OCTOBER 31. New England Obstetrical and Gynecological Society. Hotel Somerset, Boston.
NOVEMBER 10–21. Pan-Pacific Surgical Association. Page 680, issue of October 26
JULY 8–11, 1952. Thirteenth British Congress of Obstetrics and Gynaecology. Page xv, issue of December 14.

DISTRICT MEDICAL SOCIETIES

HAMPDEN

JANUARY 23. 6:30 p.m. Hotel Kimball, Springfield.

HAMPSHIRE

MARCH 7. 4:30 p.m. Cooley-Dickinson Hospital, Northampton.
MAY 2. 4:30 p.m. Annual Meeting. Jones Library, Amherst.

NORFOLK

JANUARY 23.
FEBRUARY 27.
MARCH 27.
APRIL 24.
MAY 9. Annual Dinner.
All meetings except the Annual Dinner will be held at the Boston Medical Library, 8 Fenway, at 8:00 p.m.

SUFFOLK

JANUARY 29. 8:30 p.m. Chelsea Naval Hospital.

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, JANUARY 25

THURSDAY, JANUARY 25

*9:00–10:00 a.m. Grand Rounds. Robert Breck Brigham Hospital.
*9:00–10:00 a.m. Grand Rounds. House of the Good Samaritan.
*9:00–11:00 a.m. Medical Grand Rounds. Joseph H. Pratt Diagnostic Hospital.

FRIDAY, JANUARY 26

*9:00–10:00 a.m. Medical Conference. Joseph H. Pratt Diagnostic Hospital.
*9:00 a.m.–12:00 m. Combined Medical and Surgical Staff Rounds. Peter Bent Brigham Hospital.
*10:00 a.m. Surgical Clinic. South End Health Unit, 57 East Concord Street, Boston.
*12:00 m.–1:00 p.m. Surgical Pathological Conference. Joseph H. Pratt Diagnostic Hospital.
*1:30 p.m. Tumor Clinic. Out-Patient Department, Mount Auburn Hospital, Cambridge.
*2:00–4:00 p.m. Therapeutic Conference. Joseph H. Pratt Diagnostic Hospital.
*4:00–6:00 p.m. X-ray Conference. Joseph H. Pratt Diagnostic Hospital.

SATURDAY, JANUARY 27

*9:00–10:00 a.m. Surgical Grand Rounds. Joseph H. Pratt Diagnostic Hospital.

MONDAY, JANUARY 29

*11:30 a.m.–12:15 p.m. Clinical Conference. South End Health Unit, 57 East Concord Street, Boston. Dr. Cleaveland Floyd in charge.
*12:15–1:15 p.m. Clinicopathological Conference. Main Amphitheater, Peter Bent Brigham Hospital.
5:15 p.m. Staff Meeting. Harris Hall, New England Deaconess Hospital.

TUESDAY, JANUARY 30

*9:00–10:00 a.m. Medical Conference. Joseph H. Pratt Diagnostic Hospital.
*12:15–1:15 p.m. Clinicoroentgenological Conference. Peter Bent Brigham Hospital.
*1:30–2:30 p.m. Pediatric Rounds. Burnham Memorial Hospital for Children, Massachusetts General Hospital.

WEDNESDAY, JANUARY 31

*9:00–10:00 a.m. Medical Conference. Joseph H. Pratt Diagnostic Hospital.
*11:00 a.m.–12:00 m. Medical Clinic, including Psychiatric Conferences. (Children's Medical Center.) Amphitheater, Children's Hospital.
*12:00 m. Outside speaker. Margaret Jewett Hall, Mount Auburn Hospital, Cambridge.
*12:00 m.–1:00 p.m. Clinicopathological Conference. (Children's Medical Center.) Amphitheater, Peter Bent Brigham Hospital.
*2:00–3:00 p.m. Combined Clinic of all services. (Children's Medical Center.) Amphitheater, Children's Hospital.

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Index to Advertisers

	PAGE
Abbott Laboratories	xi
Adams House	xii
American Meat Institute	iv
American Policyholders' Insurance Co.	ix
Ayerst, McKenna and Harrison, Ltd.	vi
A–Z Research Laboratory	viii
Baldpate, Inc.	xii
The Birtcher Corporation	ix
Boston Medical Supply Company	viii
Burgess & Leith	ix
Burroughs Wellcome and Company, Inc.	v
The Channing Home	xii
David Memorial Nursing Home	xii
Electroencephalographic Laboratory, Inc.	viii
Glenside, Inc.	xii
H. P. Hood and Sons	ii
Huntley Nursing Home	xii
Institute for Speech Correction, Inc.	xii
Kenmore Pharmacy, Inc.	ix
Leary Laboratory	viii
Eli Lilly and Company	Front Cover
Mead Johnson and Company	Back Cover
Perkins School	xii
Physicians Laboratory Service	viii
Ring Sanatorium	xii
U. S. Vitamin Corporation	After iv
Walker-Gordon	vii
Washingtonian Hospital	xii
Westwood Lodge, Inc.	xii
Winthrop-Stearns, Inc.	x
Wiswall Sanatorium	xii
Woodside Cottages, Inc.	xii
Wyeth, Inc.	iii

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